138014

Table 8-7

CHEMICALS DETECTED IN
UNFILTERED SURFACE WATER SAMPLES

Chemical	Frequency of Detection		MCL (value (µg/L)	Sample Quantitation Limits (µg/L)	Range of Detected Concentrations (µg/L)	Background Levels (µg/L)
	Overall	Above MCL				
Inorganics						
Aluminum	1/17	1	50 - 200 (s)	200	558	199
Arsenic*	8/17	2	50	10	3.1 - 143	ND
Barium	17/17	0	1,000	200	20.7 - 287	73.7
Calcium	17/17	NA	NA	5,000	2,860 - 69,500	6,250
Chromium*	2/17	2	100	10	135 - 153	2.4
Copper*	2/17	0	1,300 (p) 1,000 (s)	25	181 - 207	ND
Iron	17/17	17	300 (s)	100	821 - 21,800	1,950
Lead	12/17	4	5 (p)	5	1.8 - 403	2.9
Magnesium	17/17	NA	NA	5,000	1,110 - 89,700	2,560
Manganese	13/17	13	50 (s)	15	18.4 - 566	53.8
Mercury	7/17	0	2.0	0.2	.21 - .96	ND
Potassium	14/17	NA	NA	5,000	1,390 - 35,300	2,010
Sodium	16/17	NA	NA	5,000	1,900 - 744,000	8,880
Vanadium	2/17	NA	NA	50	37.9 - 43.1	ND
Zinc	9/17	0	5,000 (s)	20	3.2 - 1,690	8.2
Cyanide	1/17	0	200 (p)	10	26.4	ND

02[UZ]ZD3081:D3123/4062/14

Key at end of table.

Table 8-7 (Cont.)

Parameter	Frequency of Detection: Overall	Frequency of Detection: Above MCL	MCL (value (µg/L)	Sample Quantitation Limits (µg/L)	Range of Detected Concentrations (µg/L)	Background Levels (µg/L)
ORGANICS						
Semi-volatiles						
4-methylphenol	1/17	NA	10	10	ND	ND
Pyrene	1/17	NA	NA	10	2.0	ND
Butylbenzylphthalate	1/17	NA	NA	10	4.0	ND
Volatiles						
Dioxin/Furans*	2/3	NA	NA	0.34 - 0.63	5.8510 - 7.0080	ND

02[UZ]ZD3081:D3123/4062/14

Note: Dioxin equivalent factors were reported in unfiltered wastewater samples only. They are not reported here because there is no complete exposure pathway.

Key:

* = Chemical of potential concern.
ND = Not detected.
NA = Not available.
(p) = Proposed.
(s) = Secondary MCL based on aesthetic factors.

Source: Ecology and Environment, Inc. 1991.

Table 8-8

CHEMICALS DETECTED IN SEDIMENT SOIL

Chemical	Frequency of Detection	Range of Sample Quantitation Limits (µg/kg)	Range of Detected Concentrations (µg/kg)	Background Levels (µg/kg)
Metals				
Aluminum	18/18	0.471 - 1.205	400,000 - 17,200,000	8,700,000
Antimony	2/18	0.141 - 0.361	3,000 - 9,500	ND
Arsenic*	16/18	0.024 - 0.060	1,600 - 72,000	2,200
Barium	16/18	0.471 - 1.205	9,000 - 143,000	35,300
Beryllium	1/18	0.012 - 0.030	650	ND
Calcium	18/18	11.779 - 30.120	242,000 - 52,900,000	461,000
Chromium*	18/18	0.024 - 0.060	2,800 - 39,000	11,500
Cobalt	3/18	0.118 - 0.301	2,600 - 7,400	ND
Copper*	13/18	0.059 - 0.151	3,400 - 17,100	6,300
Iron	18/18	0.236 - 0.602	819,000 - 42,700,000	5,060,000
Lead	18/18	0.012 - 0.030	7,600 - 56,200	24,200
Magnesium	16/18	11.779 - 30.120	119,000 - 2,440,000	690,000
Manganese	18/18	0.035 - 0.090	4,500 - 628,000	17,000
Mercury	3/18	0.0005 - 0.0012	210 - 260	ND
Nickel	7/18	0.094 - 0.241	5,000 - 39,100	ND
Potassium	12/18	11.779 - 30.120	139,000 - 1,160,000	695,000
Sodium	18/18	11.779 - 30.120	29,100 - 1,260,000	313,000
Vanadium	14/18	0.118 - 0.301	3,300 - 60,000	13,700
Zinc	18/18	0.047 - 0.120	13,800 - 107,000	18,300
Cr (VI)*	14/19	10	10 - 370.1	107
Semi-Volatiles				
4-Methylphenol	1/17	3.630 - 25.410	110	ND
Naphthalene	2/17	3.630 - 25.410	230 - 5,300	ND
2-Methyl-naphthalene	1/17	3.630 - 25.410	12,000	ND
Dibenzofuran	1/17	3.630 - 25.410	710	ND
Di-n-butyl phthalate	1/17	3,630 - 25,410	280	ND
Pentachlorophenol*	4/20	17.600 - 123.200	1,200 - 230,000	ND

02[UZ]ZD3081:D3123/4066/15

Key at end of table.

Table 8-8 (Cont.)

Chemical	Frequency of Detection	Range of Sample Quantitation Limits (µg/kg)	Range of Detected Concentrations (µg/kg)	Background Levels (µg/kg)
Semi-Volatiles (Cont.)				
Phenanthrene	2/17	3.360 - 25.410	300 - 5,700	ND
Fluoranthene	2/17	3.360 - 25.410	140 - 340	ND
Benzo(a)-anthracene	2/17	3.630 - 25.410	180 - 200	ND
Chrysene	2/17	3.630 - 25.410	200 - 210	ND
bis(2-Ethyl-hexyl)phthalate	8/17	3.630 - 25.410	39 - 620	ND
Benzo(b)-fluoranthene	2/17	3.630 - 25.410	260 - 290	ND
Indeno(1,2,3-cd) pyrene	1/17	3.630 - 25.410	100	ND
Fluorene	1/17	3.630 - 25.410	2,600	ND
Benzo(g,h,i)-perylene	1/17	3.630 - 25.410	130	ND
Volatiles				
Acetone	3/17	0.120 - 0.600	120 - 210	100
Carbon disulfide	1/17	0.060 - 0.300	5	ND
2-Butanone	2/17	0.120 - 0.600	25 - 41	10
Toluene	5/17	0.060 - 0.300	5 - 60	ND
Ethylbenzene	2/17	0.060 - 0.300	10 - 140	ND
Styrene	1/17	0.060 - 0.300	5	ND
Xylenes (total)	4/17	0.060 - 0.300	5 - 290	ND
Others				
2378-TCDD	1/11		0.570	ND
Dioxin/furans*	11/11	0.078 - 0.57	0.0010 - 15.3050 (TEFs)	ND (TEFs)
TPH	17/17	75,000	8,620 - 797,000	8,620

02[UZ]ZD3081:D3123/4066/15

Key:

*Dioxin/furans results are indicated in TEFs.

NA = Not available.
ND = Not detected.

Source: Ecology and Environment, Inc. 1991.

Table 8-9

CHEMICALS DETECTED IN FILTERED
GROUNDWATER SAMPLES

Chemical	Frequency of Detection: Overall	Frequency of Detection: Above MCL	MCL (µg/L)	Sample Quantitation Limits (µg/L)	Range of Detected Concentrations (µg/L)	Background Levels (µg/L)
Metals						
Aluminum	5/18	3	50 - 200 (s)	200	45.9 - 1,180	256
Arsenic*	4/24	0	50	10	3.0 - 49	4
Barium	14/18	0	1,000	200	13.4 - 76.4	42.6
Calcium	18/18	NA	NA	5,000	11,400 - 153,000	9,010 - 38,200
Chromium*	3/24	0	100	10	7.7 - 27.8	ND
Copper*	2/24	0	1,300 (p) 1,000 (s)	25	8.8 - 11.2	ND
Iron	5/18	4	300 (s)	100	5.4 - 8,580	31.2 - 35.6
Lead	3/18	1	5 (p)	5	1.1 - 6.5	ND
Magnesium	17/18	NA	NA	5,000	942 - 6,370	768 - 4,240
Manganese	16/18	13	50 (s)	15	8.6 - 841	24.6
Potassium	17/18	NA	NA	5,000	1,180 - 27,400	6,660 - 21,800
Selenium	1/18	0	10	5	2.3	ND
Sodium	16/18	NA	NA	5,000	5,840 - 38,500	13,100 - 35,600
Vanadium	1/18	NA	NA	50	5.1	ND
Zinc	9/18	0	5,000 (s)	20	13.0 - 268	30.4 - 35.3

02[UZ]ZD3081:D3123/4191/16

Key:

* = Chemical of potential concern.
ND = Not detected.
NA = Not available.
(p) = Proposed.
(s) = Secondary MCL based on aesthetic factors.

Source: Ecology and Environment, Inc. 1991.

Table 8-10

CHEMICALS DETECTED IN
UNFILTERED GROUNDWATER SAMPLES

Chemical	Frequency of Detection: Overall	Frequency of Detection: Above MCL	MCL (µg/L)	Sample Quantitation Limits (µg/L)	Range of Detected Concentrations (µg/L)	Background Levels (µg/L)
INORGANICS						
Aluminum	17/18	17	50 - 200 (s)	200	157 - 91,400	957 - 50,600
Arsenic*	16/24	1	50	10	2 - 82	6.1 - 8.9
Barium	16/18	0	1,000	200	4.9 - 384	4.9 - 177
Beryllium	6/18	4	1 (p)	5	0.76 - 13.4	ND
Cadmium	4/18	4	5	5	5.3 - 29.5	ND
Calcium	17/18	NA	NA	5,000	24,400 - 367,000	29,300 - 48,800
Chromium*	12/24	7	100	10	12.5 - 238	55.2
Cobalt	2/18	NA	NA	50	9.61 - 14.5	ND
Copper*	15/24	0	1,300 (p) 1,000 (s)	25	9.2 - 493	20.5
Iron	16/18	16	300 (s)	100	210 - 66,500	1,410 - 33,400
Lead	16/18	15	50 5 (p)	5	2.7 - 318	1.3 - 51.2
Magnesium	17/18	NA	NA	5,000	1,310 - 31,400	1,440 - 6,290
Manganese	17/18	17	50 (s)	15	10 - 1,830	13.7 - 104

02[UZ]ZD3081:D3123/4063/17

Key at end of table.

Table 8-10 (Cont.)

Chemical	Frequency of Detection: Overall	Frequency of Detection: Above MCL	MCL (µg/L)	Sample Quantitation Limits (µg/L)	Range of Detected Concentrations (µg/L)	Background Levels (µg/L)
INORGANICS (Cont.)						
Mercury	3/18	0	2	0.2	.17 - 1.5	0.37
Potassium	18/18	NA	NA	5,000	4,010 - 172,000	9,980 - 24,900
Silver	2/18	0	50 100 (s)	10	9.1 - 9.25	ND
Sodium	17/18	NA	NA	5,000	4,810 - 42,500	13,900 - 40,300
Vanadium	11/18	NA	NA	50	14 - 232	91.7
Zinc	15/18	0	5,000 (s)	20	74.3 - 741	151 - 169
Cyanide	4/18	0	200 (p)	10	6.0 - 155	16.7 - 19.4
Chromium (VI)*	1/9	0	50	10 - 20	12	ND
ORGANICS						
Semi-Volatiles						
Isophorone	1/18	NA	NA	10	1	ND
Naphthlene	1/18	NA	NA	10	3.0	ND
2-Methylnaphthalene	1/18	NA	NA	10	2.0	ND
Acenaphthalene	2/18	NA	NA	10	3.0 - 4.0	ND

02[UZ]ZD3081:D3123/4063/17

Key at end of table.

Table 8-10 (Cont.)

Chemical	Frequency of Detection: Overall	Frequency of Detection: Above MCL	MCL (µg/L)	Sample Quantitation Limits (µg/L)	Range of Detected Concentrations (µg/L)	Background Levels (µg/L)
Semi-Volatiles (Cont.)						
Pentachlorophenol*	11/31	11	1 (p)	50 - 5,000	1.0 - 19,000	ND
Phenanthrene	1/18	NA	NA	10 - 1,000	5.0	ND
Di-n-octylphthalate	2/18	NA	NA	10	1.0	ND
bis(2-Ethylhexyl) phthalate	3/18	1	4 (p)	10	3.0 - 5.0	2.0
Volatiles						
Acetone	1/18	NA	NA	10	5.0	8
Toluene	1/18	0	1,000 2,000 (p)	5	6.0	ND
Ethylbenzene	1/18	0	700	5	5	ND
Total Xylenes	1/18	0	10,000	5	49	ND
OTHERS						
TPH	1/25	NA	NA	1,000	1,400	ND

02[UZ]ZD3081:D3123/4063/17

Key:

* = Chemicals of potential concern.
(s) = Secondary.
(p) = Proposed.
NA = Not available.
ND = Not detected.

Source: Ecology and Environment, Inc. 1991.

Table 8-11

SUMMARY OF CHEMICALS OF POTENTIAL CONCERN

Chemical	Concentrations				
	Surface Soil (µg/kg)	Boring (µg/kg)	Sediment Soil (µg/kg)	Surface Water (µg/L)	Groundwater (µg/L)
Arsenic	930 - 266,000	710 - 25,400	1,600 - 72,000	3.1 - 143 4.4 - 55.9*	2.0 - 82 3.0 - 49*
Chromium (total)	1,400 - 252,000	1,700 - 47,500	2,800 - 39,000	135 - 153 9.5*	12.5 - 238 7.7 - 27.8*
Copper	1,800 - 158,000	930 - 32,900	3,400 - 17,100	181 - 207 ND*	9.2 - 493 8.8 - 11.2*
Pentachlorophenol	21 - 5,100	45 - 1,900,000	1,200 - 230,000	ND NA*	1.0 - 19,000 NA*
Dioxin/furans (TEFs)	0.0110 - 3.2490	0.0010 - 20.5690	0.0010 - 15.3050	5.8510 - 7.0080 NA*	ND NA*
Chromium (VI)	14.5 - 1,038.6	21.5 - 1,325.2	10.0 - 370.1	ND ND*	12 ND*

02[UZ]ZD3081:D3123/4064/18

Key:

* = Filtered water sample.
NA = Not available.
ND = Not detected.

Source: Ecology and Environment, Inc. 1991.

were not included where the frequencies of (parameter) detection and overall ranges of analyzed parameter concentrations in the tables found in Section 8 were listed. Therefore some variations in frequencies and ranges will be observed when comparing tables in Section 8 to the more general tables found in Section 5.

The chemicals of potential concern identified during this RI include:

- Arsenic, copper, chromium, pentachlorophenol, and dioxin/furans in soils in the wood treatment and conical burn pit areas;
- Arsenic, copper, chromium, and dioxin/furans in surface soils widely distributed on the Saunders Supply Company property;
- Dioxins and pentachlorophenol in sediments in the wastewater treatment pond;
- Pentachlorophenol in groundwater near the conical burn pit and wood treatment areas; and
- Dioxin/furans, arsenic, chromium, and copper in water and sediments in the runoff water catch basins.

Other than in the water samples taken from the run-off water catch basins (see discussion in Section 5), no contaminants related to wood treatment operations were detected at concentrations significantly higher than local background values in surface waters.

8.3 EXPOSURE ASSESSMENT

Exposure assessment procedure involves evaluating study area characteristics, identifying potentially exposed populations, understanding fate and transport of contaminants, and identifying potential exposure pathways. The study area history, setting, nature and extent of contamination, and fate and transport of contaminants have been described fully in Sections 1, 2, 4, 5, and 6 of this report. Below is a brief summary of the information relevant to the risk assessment. Based on these conditions, the exposure estimates are presented, followed by a discussion of the exposure estimate limitations.

8.3.1 Characterization of Site Setting

The Saunders property is located within the village of Chuckatuck, a rural area of the consolidated independent City of Suffolk. The City of Suffolk encompasses 430 square miles of urban, suburban, and rural areas. In 1988, the City of Suffolk had an estimated population of 52,200 persons (U.S. Department of Commerce, Bureau of the Census 1989). According to EPA's Geographical Exposure Modeling System (GEMS), 4,277 persons live within 2 miles of the Saunders property (based on 1983 population estimates) and 4,930 persons live within 4 miles of the site.

Land in the City of Suffolk is primarily undeveloped. Approximately 70% is forested, open lands, or lakes. Agriculture represents 25% of the land use; residential use is 3%; commercial uses are 1%; and manufacturing less than 0.5% (Vacalis 1990). Land use within 1 mile of the Saunders property is predominantly agricultural and rural residential.

Saunders Supply Company is located along State Route 10/32 (Godwin Boulevard) near the intersection with State Route 125 (Kings Highway). State Route 10/32 is the eastern boundary of the property. At this intersection, there is a concentration of residential and commercial establishments, which decrease in density less than 0.5 mile from the intersection. A fire station is located to the southeast of this intersection.

The northern edge of the Saunders property abuts several residences and a commercial nursery. Residences also abut the southern edge of the property, located along Crumpler Lane, and the southwestern edge of the property. At the corner of Crumpler Lane and Godwin Boulevard is a gas station. To the west of the property is an intermittent stream, which crosses the nursery and flows into Godwin's Millpond, a municipal drinking water source and potential fishing water body, located approximately 500 feet north of the Saunders property. The Saunders property is underlain by an upper unconsolidated aquifer and a lower confined aquifer. The upper unconsolidated Columbian aquifer discharges primarily to the intermittent stream and Godwin's Millpond. The lower confined Yorktown aquifer discharges primarily to Godwin's Millpond.

Godwin's Mill pond, Lone Star Lakes and the G. Robert House fluoridation well are the primary water supplies for the City of Suffolk. Water supply wells formerly used by the village of Chuckatuck have been shut down within the past year. Any homes originally serviced by the former water supply wells currently receive their water from the City of Suffolk. All other residences in the village of Chuckatuck are assumed to be on individual wells. Residences and establishments located along Godwin Boulevard, however, can tie into the City of Suffolk's water main, which parallels Godwin Boulevard.

8.3.2 Potentially Exposed Populations

The identity of potentially exposed populations can best be understood by considering the nature and location of contaminants found on the Saunders property. Contaminants related to former wood treatment operations were found in soils throughout the Saunders property in sediments in the wastewater pond, and in groundwater. Exposures to soil or sediment contaminants are most likely to occur on the Saunders property. Groundwater contaminants could migrate, potentially exposing anyone who uses the groundwater as a drinking water source. Currently the groundwater at the site is not used directly for residential purposes, but flows to Godwin's Millpond, which is a drinking water source.

Based on the characterization and current land usage in the area, the current potentially exposed populations include the following groups:

- Employees of Saunders working at the Saunders Supply Company and potentially exposed to contaminated site soils; and
- People drinking water from Godwin's Millpond if and when it becomes contaminated by groundwater.

Since the Saunders property is currently surrounded by residential areas on three sides, it is possible that the Saunders property itself could be converted to residential usage at some time in the future. If this were to occur without any remedial measures taken at the site, future residents could potentially be exposed to contaminants by several exposure routes.

8.3.3 Sources and Receiving Media

The sources and receiving media of environmental contamination associated with the Saunders Supply Company resulted primarily from former wood treatment and waste disposal practices. Beginning in 1964, wood preservation activities have been performed by the Saunders Supply company on the Saunders property. PCP solutions were used from approximately 1964 to 1984; CCA solutions from 1974 to the present.

Previous activity on the Saunders property included the use of an unlined oil/water separation pond (the former earthen berm area) located southeast of the existing wastewater pond as an oil/water separator. A crust-like residue, which formed on the pond surface, was occasionally burned as a training exercise for the local fire department. Water from the unlined pond was discharged to the wastewater pond. Water from the wastewater pond was occasionally discharged to the intermittent stream. Water from the wastewater pond was also used for the CCA process. Since the conversion to the CCA process, completed in 1984, wastewater has not been generated. (CCA is a process that is a net water user.)

Sludges removed during annual maintenance of the PCP treatment cylinders or associated oil/water separators were used on the roads and/or around the lumber piles to control dust and weeds from approximately 1966 through 1981. In 1969 a conical burner was used to incinerate some of the sludges, lumber scraps, and sawdust. The conical burner ceased operations in 1974 and has since been removed. Off-site disposal of sludges generated by the PCP process took place from 1981 through 1985.

During active treatment operations on the site, treated wood has been allowed to dry on the Saunders property. Prior to 1984, treated wood was placed on pallets located directly on the ground in the southern portion of the site. Since 1984 the wood has been air-dried on a concrete drip pad, which collects excess chemicals.

An initial remedial action was completed in 1984 to excavate the soils beneath the conical burner and to install a recovery well. Despite these activities soils in the vicinity of the conical burner and wood treating area remain among the most heavily contaminated with dioxin/furans and PCP, and groundwater immediately downgradient of the

pit is contaminated with PCP. Shallow groundwater flows to the intermittent stream; deep groundwater flows below the nursery property to Godwin's Millpond. Dioxin/furans were also found in high concentrations in sediments of the existing wastewater pond, and PCP, dioxin/furans, arsenic, chromium, and copper are widely distributed in site soils.

8.3.4 Fate and Transport of Contaminants in the Environment

A variety of environmental and chemical-specific factors influence the fate and transport of contaminants in the environment, as was discussed in detail in Section 6. Soils in the form of dust are raised into the breathing zone by facility and vehicle traffic. These surficial soils are also moved off the Saunders property by surface runoff and ultimately can reach the adjacent surface waters. (This transport pathway is discussed in Section 9, Ecological Assessment.) Both the shallow and deep water-bearing zones beneath the site also offer transport pathways for contaminants to either enter into one of the adjacent surface waters or remain in the groundwater system. The mobility of the contaminants in the various environmental media will be controlled by their physical and chemical properties. These properties are important determinants that directly reflect the exposure potential for humans and environmental receptors and, as noted earlier, are discussed in detail in Section 6.

8.3.5 Exposure Pathways

Based on the RI, there appear to be five primary areas of contamination associated with the Saunders Supply Company:

- Soils in the vicinity of the former location of the conical burn pit and earthen separation pond;
- Sediments in the existing wastewater pond;
- Surface soils widely distributed on the Saunders property and portion of the adjoining Kelly property;
- A groundwater plume adjacent to the conical burn pit and wood treating process area; and
- Water/sediment in runoff water catch basins.

The contaminants of greatest concern with respect to potential current exposures are those in the surface soils. Exposure to soil contaminants could potentially occur by three exposure routes:

- o Direct contact with contaminated soil;
- o Ingestion of soil by hand-to-mouth contact; and
- o Inhalation of airborne particles of soil.

Exposures by these routes are most likely to occur on the Saunders property, and currently Saunders Supply Company employees are the main receptors. If the Saunders property was converted to residential use, existing subsurface soils could become surface soils as a result of regrading the site or excavation for basements. Future residents could be exposed to contaminants in both surface and subsurface soil by the same three exposure routes.

Currently exposure to contaminants in sediment at the bottom of the wastewater pond is very unlikely. If, in the future, the Saunders property was developed for residential use, it is conceivable that the pond could be drained, thereby exposing the sediments. Future residents could potentially be exposed by the soil exposure pathways described above.

Currently exposure to groundwater contamination, primarily PCP, is also very unlikely. Groundwater at the Saunders property is not used as a water supply source and, although the groundwater flows to Godwin's Millpond, which is a water supply source, PCP has not been detected there. If the site was converted for residential use with groundwater as the drinking water source, future residents could potentially be exposed to PCP by the following pathways:

- o Ingestion of drinking water;
- o Dermal contact during showering; and
- o Inhalation of vapors during showering.

The contaminants found in the water in the runoff water catch basins are thought to be associated with the sediment (see discussion,

Section 5) and immobile. The catch basins are covered with grates, which effectively prevent exposures to Saunders Supply Company employees and others.

Additionally, sampling downstream of the catch basins (CE-1) provided evidence that the sediments posed no risk to downstream receptors. Since the sediments in the catch basins appear to pose no risk under current site conditions for normal site activities, the sediments in the catch basins were not considered in the baseline risk assessment.

8.3.6 Exposure Scenarios

Three scenarios, encompassing the greatest potential exposure pathways, are evaluated in this document. They are:

- Scenario 1: Worker Exposure to Soil Contaminants;
- Scenario 2: Hypothetical Future Residential Exposure to Soil Contaminants; and
- Scenario 3: Residential Groundwater Usage Exposures

Scenario 1 addresses outdoor exposure to adult workers under existing conditions. Given that soils are contaminated with metals and relatively nonvolatile organic compounds, the worker exposure scenario addresses exposure due to ingestion and dermal absorption of contaminants in soil, and inhalation of soil contaminants entrained in airborne particulates.

Scenario 2 addresses soil-related exposures that could occur if the Saunders property were to be converted to residential use at some time in the future. In this scenario, the same three exposure pathways (*ingestion, dermal absorption, and inhalation*) will be evaluated for adult males and females, teenagers, adolescents, and young children.

Scenario 3 addresses potential exposures to pentachlorophenol that could occur as the result of future residential use of groundwater from the site. The exposure pathways evaluated are ingestion of groundwater, dermal contact during showering, and inhalation of volatilized PCP during showering. Because of the extremely limited data available for the Yorktown aquifer and the limited understanding of the potential

communication between the Columbian and Yorktown aquifers, and general groundwater migration rates and patterns in the vicinity of the site, the groundwater contaminant concentrations used to make exposure estimates for this scenario should not be viewed as representative concentrations that might occur at actual exposure points. The exposure estimates for this scenario are not intended to represent actual potential exposures, but only to provide some reference points for evaluating the risks future groundwater use might pose. For simplicity, only adult male receptors were evaluated in this scenario.

8.3.7 Exposure Concentrations

As per USEPA guidance, the upper 95% confidence limits on the arithmetic averages of surface soil concentrations were used to estimate exposures under the current Saunders Supply Company worker exposure scenario. Since soil would be excavated prior to residential construction, upper 95% confidence limits for all surface soil, wastewater pond sediment, and soil boring samples were used in estimating exposures in the future site-use residential scenario. Table 8-12 presents the soil concentrations used in exposure estimation for these two scenarios.

Only the deeper Yorktown aquifer is used as a drinking water source in the vicinity of the Saunders property; however, there appears to be hydraulic communication between the upper Columbian water-bearing zone and the Yorktown aquifer beneath the Saunders property, probably by way of open boreholes through the intervening clay layer. Thus, the more contaminated water in the Columbian aquifer could migrate down into the Yorktown aquifer and increase the contaminant concentration in that unit.

Therefore, two exposure estimates were made for the residential groundwater use scenario, one for groundwater from the lower aquifer, and another for groundwater from the more contaminated upper aquifer. For the lower aquifer, there were only two usable data values, so the maximum concentration was used as the PCP exposure concentration. For the upper aquifer, the upper 95% confidence limit, based on six data values, was used. The data used is summarized in Table 8-13.

Table 8-12

CONCENTRATIONS OF CONTAMINANTS USED
IN ESTIMATING EXPOSURE
TO SITE SOIL

Contaminant	Soil	
	Current Site Use (mg/kg)*	Future Site Use (mg/kg)**
Arsenic	106	16
Chromium (III)	119	25
Chromium (VI)	0.46	0.32
Copper	87	18
Pentachlorophenol	1.6	62.6
2,3,7,8-TCDD Equivalents	0.0019	0.00241

02[UZ]ZD3081:D3123/4165/35

Key:

*Upper 95% confidence limit on mean concentration in surface soils collected on the Saunders property only.

**Upper 95% confidence limit on mean concentration in all surface soil and soil boring samples.

Source: Ecology and Environment, Inc., 1991.

Table 8-13

CONCENTRATIONS OF PCP
USED IN ESTIMATING EXPOSURE
TO SITE GROUNDWATER

Sample Number	PCP (µg/L)
Lower Aquifer	
MW-8-D	160
MW-8-II	11
Maximum Concentration	160
Upper Aquifer	
MW-4-II	19,000
MW-4-S	15,000
MW-7-II	25
MW-7-S	10
MW-15-II	240
MW-16-II	4,100
Upper 95% Confidence Limit	12,074

[UZ]ZD3081:D3123/5040/34

Source: Ecology and Environment, Inc. 1991.

8.3.8 Exposure Estimation Methods

The scenarios described above were used to estimate potential exposures using equations of the form depicted in the following equation:

$$\text{Intake} = \frac{(\text{Concentration})\ (\text{Contact Rate})\ (\text{Exposure Frequency})\ (\text{Exposure Duration})\ (\text{Absorption Fraction})}{(\text{Body Weight})\ (\text{Averaging Time})}$$

As presented in the equation, exposure, or intake, is directly proportional to the product of contaminant concentration, contact rate, frequency of exposure, exposure duration, and absorption fraction divided by the product of body weight and averaging time.

Given concern for both short- and long-term public health risks, such equations enable estimation of both lifetime average daily intakes (LADIs) used in the evaluation of potential carcinogenic risks, and chronic daily intakes (CDIs) and subchronic daily intakes (SCDIs) calculated for pathway-specific exposure periods, which are used in the evaluation of noncarcinogenic risks.

This section integrates populations, wood treating activities, and exposure pathways into exposure scenarios, enabling evaluation of human health risks.

Tables 8-14 through 8-22 present pathway-specific equations--the parameter values used and the references or rationale for selecting the values used in estimating the LADIs and CDIs. Common to all the scenarios are fixed-receptor body weights and a common way to estimate averaging times. Body weight for typical adult male receptors was taken as 70 kilograms; for a 4-year old (a typical 2- to 6-year old), 16 kilograms; for a 9-year old (a typical 6- to 12-year old), 31 kilograms; for a 15-year old (a typical 12- to 18-year old), 55 kilograms; and for an adult female, 58 kilograms, based upon reported body weights (EPA 1989b). Typical body weights for workers were also taken as 70 kg (EPA 1989b). Consistent with the EPA-recommended approach, averaging time for carcinogenic effects was taken as 70 years, the assumed length of an average lifetime, and for noncarcinogenic effects, the product of the exposure duration times 365 days/year (EPA 1989a) was used.

Table 8-14

SCENARIO 1
WORKER EXPOSURE: INGESTION OF CHEMICALS IN SOIL

Equation:

$$\text{Intake (mg/kg-day)} = \frac{\text{CS} \times \text{INGR} \times \text{CF} \times \text{FI} \times \text{EF} \times \text{ED}}{\text{BW} \times \text{AT}}$$

where:

CS = Chemical Concentration in Soil (mg/kg)
INGR = Ingestion Rate (mg soil/day)
CF = Conversion Factor (10^{-6} kg/mg)
FI = Fraction Ingested from Contaminated Source (unitless)
EF = Exposure Frequency (days/years)
ED = Exposure Duration (years)
BW = Body Weight (kg)
AT = Averaging Time (period over which exposure is averaged - days)

Variable	Case	Receptor	Value (Rationale/Source)
CS	RME	Adult	Upper 95% confidence limit on concentrations in surface soil
INGR	RME	Adult	100 mg/day (age groups greater than 6 years old; EPA 1989c)
FI	RME	Adult	1.0 (assumed)
EF	RME	Adult	250 days/year
ED	RME	Adult	40 years (EPA 1989b)
BW	RME	Adult	70 kg (average; EPA 1989b)
AT	RME	Adult	Pathway-specific period of exposure for noncarcinogenic effects (i.e., ED x 365 days/year), and 70 year lifetime for carcinogenic effects (i.e., 70 years x 365 days/year)

02[UZ]ZD3081:D3123/3954/20

Source: Ecology and Environment, Inc., 1991.

Table 8-15

SCENARIO 1
WORKER EXPOSURE: DERMAL CONTACT WITH CHEMICALS IN SOIL

Equation:

$$\text{Absorbed Dose (mg/kg-day)} = \frac{CS \times ABS \times CF \times SA \times AF \times EF \times ED}{BW \times AT}$$

where:

CS = Chemical Concentration in Soil (mg/kg)
ABS = Fraction Absorbed (Unitless)
CF = Conversion Factor (10^{-6} kg/mg)
SA = Skin Surface Area Available for Contact (cm^2/event)
AF = Soil to Skin Adherence Factor (mg/cm^2)
EF = Exposure Frequency (events/year)
ED = Exposure Duration (years)
BW = Body Weight (kg)
AT = Averaging Time (period over which exposure is averaged - days)

Variable	Case	Receptor	Value (Rationale/Source)
CS	RME	Adult	Upper 95% confidence limit on concentrations in surface soil
ABS	RME	Adult	Chemical-specific value
SA	RME	Adult	800 cm^2 (hands; surface area; EPA 1989b)
AF	RME	Adult	1.45 mg/cm^2 (EPA 1989b)
EF	RME	Adult	250 days/year (5 days/week for 50 weeks)
ED	RME	Adult	40 years (EPA 1989b)
BW	RME	Adult	70 kg (average; EPA 1989b)
AT	RME	Adult	Pathway-specific period of exposure for noncarcinogenic effects (i.e., ED x 365 days/year), and 70 year lifetime for carcinogenic effects (i.e., 70 years x 365 days/year)

02[UZ]ZD3081:D3123/3955/19

Source: Ecology and Environment, Inc., 1991.

Table 8-16

SCENARIO 1
WORKER EXPOSURE: INHALATION AIRBORNE SOIL PARTICULATE

Equation:

$$\text{Intake (mg/kg-day)} = \frac{CS \times CP \times CF \times IR \times ET \times EF \times ED}{BW \times AT}$$

where:

CS = Contaminant Concentration in Soil (mg/kg)
CP = Particulate Concentration in Air (mg/m^3)
CF = Unit Conversion Factor (10^{-6} kg/mg)
IR = Inhalation Rate (m^3/hour)
ET = Exposure Time (hours/day)
EF = Exposure Frequency (days/year)
ED = Exposure Duration (years)
BW = Body Weight (kg)
AT = Averaging Time (period over which exposure is averaged - days)

Variable	Case	Receptor	Value (Rationale/Source)
CP	RME	Adult	Site-specific value
CS	RME	Adult	Upper 95% confidence limit on concentrations in soil
IR	RME	Adult	2.5 m^3/hr (moderate activity; EPA 1989b)
ET	RME	Adult	8 hours/day
EF	RME	Adult	250 days/year (5 days/week for 50 weeks)
ED	RME	Adult	40 years (EPA 1989b)
BW	RME	Adult	70 kg (adult, average; EPA 1989b)
AT	RME	Adult	Pathway-specific period of exposure for noncarcinogenic effects (i.e., ED x 365 days/year) and 70 year lifetime for carcinogenic effects (i.e., 70 years x 365 days/year)

02[UZ]ZD3081:D3123/3952/20

Source: Ecology and Environment, Inc. 1991.

Table 8-17

SCENARIO 2
RESIDENTIAL EXPOSURE: INGESTION OF CHEMICALS IN SOIL

Equation:

$$\text{Intake (mg/kg-day)} = \frac{CS \times INGR \times CF \times FI \times EF \times ED}{BW \times AT}$$

where:

CS = Chemical Concentration in Soil (mg/kg)
INGR = Ingestion Rate (mg soil/day)
CF = Conversion Factor (10^{-6} kg/mg)
FI = Fraction Ingested from Contaminated Source (unitless)
EF = Exposure Frequency (days/years)
ED = Exposure Duration (years)
BW = Body Weight (kg)
AT = Averaging Time (period over which exposure is averaged - days)

Variable	Case	Receptor	Value (Rationale/Source)
CS	RME	All	Upper 95% confidence limit on concentrations in soil
INGR	RME	Adults Teenager Adolescent	100 mg/day (age groups greater than 6 years old; EPA 1989c)
	RME	Child	200 mg/day (children 1 through 6 years old; EPA 1989c)
FI	RME	All	1.0 (assumed)
EF	RME	All	365 days/year
ED	RME	Adults	30 years (national median time (90th percentile) at one residence; EPA 1989b)
		Others	Entire period in age bracket, assumed spent at one residence (6 years for teenagers and adolescents, 5 years for children)
BW	RME	All	Median body weight for each respective age group (70 kg adult male, 58 kg adult female, 55 kg teenager, 31 kg adolescent, 16 kg child; EPA 1989b)
AT	RME	All	Pathway-specific period of exposure for noncarcinogenic effects (i.e., ED x 365 days/year), and 70 year lifetime for carcinogenic effects (i.e., 70 years x 365 days/year)

02[UZ]ZD3081:D3123/3953/19

Source: Ecology and Environment, Inc. 1991.

Table 8-18

SCENARIO 2
RESIDENTIAL EXPOSURE: DERMAL CONTACT WITH CHEMICALS IN SOIL

Equation:

$$\text{Absorbed Dose (mg/kg-day)} = \frac{CS \times ABS \times CF \times SA \times AF \times EF \times ED}{BW \times AT}$$

where:

CS = Chemical Concentration in Soil (mg/kg)
ABS = Fraction Absorbed (Unitless)
CF = Conversion Factor (10^{-6} kg/mg)
SA = Skin Surface Area Available for Contact (cm^2/event)
AF = Soil to Skin Adherence Factor (mg/cm^2)
EF = Exposure Frequency (events/year)
ED = Exposure Duration (years)
BW = Body Weight (kg)
AT = Averaging Time (period over which exposure is averaged - days)

Variable	Case	Receptors	Value (Rationale/Source)
CS	RME	All	Upper 95% confidence limit on concentrations in soil
ABS	RME	All	Chemical-specific value
SA	RME	Adults	1,600 cm^2 (hands and one-third arms; surface area; EPA 1989b)
	RME	Teenager	1,600 cm^2 (hands and one-third arms; surface areas; EPA 1985)
	RME	Adolescent	2,000 cm^2 (hands and one-third arms and legs; surface areas; EPA 1989b)
	RME	Child	1,800 cm^2 (hands and one-half arms and legs; surface areas; EPA 1989b)
AF	RME	All	1.45 mg/cm^2 (EPA 1989b)
EF	RME	All	365 days/year
ED	RME	Adults	30 years (national upper bound time (90th percentile) at one residence; EPA 1989b)
		Others	Entire duration of each respective age group (6 years for teenagers and adolescents, 5 years for children)
BW	RME	All	Median body weights for each respective age group (70 kg adult male, 58 kg adult female, 55 kg teenager, 31 kg adolescent, 16 kg child; EPA 1989b)
AT	RME	All	Pathway-specific period of exposure for noncarcinogenic effects (i.e., ED x 365 days/year), and 70 year lifetime for carcinogenic effects (i.e., 70 years x 365 days/year)

02[UZ]ZD3081:D3123/3949/20

Source: Ecology and Environment, Inc., 1991.

Table 8-19

SCENARIO 2
RESIDENTIAL EXPOSURE: INHALATION AIRBORNE PARTICULATE

Equation:

$$\text{Intake (mg/kg-day)} = \frac{CS \times CP \times CF \times IR \times ET \times EF \times ED}{BW \times AT}$$

where:

CS = Contaminant Concentration in Soil (mg/kg)
CP = Particulate Concentration in Air (mg/m^3)
CF = Unit Conversion Factor (10^{-6} kg/mg)
IR = Inhalation Rate (m^3/hour)
ET = Exposure Time (hours/day)
EF = Exposure Frequency (days/year)
ED = Exposure Duration (years)
BW = Body Weight (kg)
AT = Averaging Time (period over which exposure is averaged - days)

Variable	Case	Receptors	Value (Rationale/Source)
CP	RME	All	Site-specific value
CS	RME	All	Maximum concentration in soil (mg/kg)
IR	RME	Adult male	1.2 m^3/hr (Recommended value for adult; EPA 1989b)
		Adult female	1.2 m^3/hr (Recommended value for adult; EPA 1989b)
		Teenager	1.2 m^3/hr (In absence of age-specific data, value is based on adult rate; EPA 1989b)
		Adolescent	1.5 m^3/hr (Consistent with EPA guidance for adults; 1.5 times light activity rate for 10-year old; EPA 1989b)
		Child	1.2 m^3/hr (Consistent with EPA guidance for adults; 1.5 times light activity rate for a 6-year old; EPA 1989b)
ET	RME	All	16 hours/day
EF	RME	All	365 days/year
ED	RME	Adults	30 years (90th percentile time at one residence) (EPA 1989b)
	RME	Others	Entire duration (entire period of life in each age group; 6 years for teenagers and adolescents, 5 years for children)
BW	RME	All	Median body weights for each respective age group (70 kg adult male, 58 kg adult female, 55 kg teenager, 31 kg adolescent, 16 kg child; EPA 1989b)

02[UZ]ZD3081:D3123/3956/20

Table 8-19 (Cont.)

Variable	Case	Receptors	Value (Rationale/Source)
AT	RME	All	Pathway-specific period of exposure for noncarcinogenic effects (i.e., ED x 365 days/year) and 70 year lifetime for carcinogenic effects (i.e., 70 years x 365 days/year)

02[UZ]ZD3081:D3123/3956/20

Source: Ecology and Environment, Inc. 1991.

Table 8-20

SCENARIO 3
RESIDENTIAL WATER USAGE: INGESTION OF CHEMICALS IN DRINKING WATER
(AND BEVERAGES MADE WITH DRINKING WATER)

Equation:

$$\text{Intake (mg/kg-day)} = \frac{CW \times INGR \times EF \times ED}{BW \times AT}$$

where:

CW = Chemical Concentration in Water (mg/liter)
INGR = Ingestion Rate (L/day)
EF = Exposure Frequency (days/year)
ED = Exposure Duration (years)
BW = Body Weight (kg)
AT = Averaging Time (period over which exposure is averaged - days)

Variable	Case	Receptor	Value (Rationale/Source)
CW	RME	Adult	Measured concentrations in ground-water
INGR	RME	Adult	2 L/day (90th percentile; EPA 1989b)
EF	RME	Adult	365 days/year
ED	RME	Adult	30 years (90th percentile time at one residence, EPA 1989b)
BW	RME	Adult	70 kg (EPA 1989b)
AT	RME	Adult	Pathway-specific period of exposure for noncarcinogenic effects (i.e., ED x 365 days/year), and 70 year lifetime for carcinogenic effects (i.e., 70 years x 365 days/year)

02[UZ]ZD3081:D3123/5041/20

Key:

RME = Reasonable Maximum Exposure.

Source: Ecology and Environment, Inc. 1991.

Table 8-21

SCENARIO 3
RESIDENTIAL WATER USAGE: DERMAL CONTACT WITH CHEMICALS DURING SHOWERING

Equation:

$$\text{Absorbed Dose (mg/kg-day)} = \frac{CW \times PC \times SA \times ET \times EF \times ED \times CF}{BW \times AT}$$

where:

CW = Chemical Concentration in Water (mg/liter)
PC = Chemical-specific Dermal Permeability Constant (cm/hr)
SA = Skin Surface Area Available for Contact (Cm^2)
ET = Exposure Time (hours/day)
EF = Exposure Frequency (days/year)
ED = Exposure Duration (years)
CF = Volumetric Conversion Factor for Water (1 liter/1000 cm^3)
BW = Body Weight (kg)
AT = Averaging Time (period over which exposure is averaged - days)

Variable	Case	Receptor	Value (Rationale/Source)
CW	RME	Adult	Measured concentrations in groundwater
PC	RME	Adult	Chemical-specific values used
SA	RME	Adult	1.8 m^2 (total body average SA for adults; EPA 1989b)
ET	RME	Adult	0.2 hour/day (12 minutes; 90th percentile; EPA 1989b)
EF	RME	Adult	365 days/year
ED	RME	Adult	30 years (90th percentile time at one residence) (EPA 1989b)
BW	RME	Adult	70 kg (EPA 1989b)
AT	RME	Adult	Pathway-specific period of exposure for noncarcinogenic effects (i.e., ED x 365 days/year), and 70 year lifetime for carcinogenic effects (i.e., 70 years x 365 days/year)

02[UZ]ZD3081:D3123/5043/20

Key:

RME = Reasonable Maximum Exposure

Source: Ecology and Environment, Inc. 1991.

Table 8-22

SCENARIO 3
RESIDENTIAL WATER USAGE: INHALATION OF AIRBORNE (VAPOR PHASE) CHEMICALS DURING SHOWERING

Equation:

$$\text{Intake (mg/kg-day)} = \frac{CA \times IR \times ET \times EF \times ED}{BW \times AT}$$

where:

CA = Contaminant Concentration in Air (mg/m^3)
IR = Inhalation Rate (m^3/hour)
ET = Exposure Time (hours/day)
EF = Exposure Frequency (days/year)
ED = Exposure Duration (years)
BW = Body Weight (kg)
AT = Averaging Time (period over which exposure is averaged - days)

Variable	Case	Receptor	Value (Rationale/Source)
CA	RME	Adult	Modeled value (based on concentrations in water)
IR	RME	Adult	0.6 m^3/hr (all age groups, EPA 1989b)
ET	RME	Adult	0.2 hour/day (12 minutes; 90th percentile; EPA 1989b)
EF	RME	Adult	365 days/year
ED	RME	Adult	30 years (90th percentile time at one residence; EPA 1989b)
BW	RME	Adult	70 kg (EPA 1989b)
AT	RME	Adult	Pathway-specific period of exposure for noncarcinogenic effects (i.e., ED x 365 days/year), and 70 year lifetime for carcinogenic effects (i.e., 70 years x 365 days/year)

02[UZ]ZD3081:D3123/5042/20

Key:

RME = Reasonable Maximum Exposure

Source: Ecology and Environment, Inc. 1991.

Scenario 1: Outdoor Worker Exposure

Key variables in the RME worker exposure scenario, summarized in Tables 8-14 through 8-16, include soil ingestion rate (INGR), exposure frequency (EF), exposure duration (ED), soil to skin adherence factor (AF), skin surface area (SA), daily exposure time (ET), and inhalation rates (IR).

The value for INGR was taken as 100 mg/day based upon EPA guidance (EPA 1989c). Exposure frequencies of 250 days/year were based upon the assumption that worker exposure would occur 5 days/week for 50 weeks/year. ED was taken as 40 years based upon EPA guidance (EPA 1989b). AF was taken as 1.45 mg/cm^2 based on EPA guidance (EPA 1989b). SA was taken as 800 cm^2, equivalent to the surface area of the hands (EPA 1989b), based on likely work activities that might result in worker exposure to soils. ET was taken as 8 hours/day to reflect the the possibility in the context of an RME scenario that exposure could occur over the entire period of a typical work day. IR was taken as 2.5 m^3/day based upon moderate energy worker activities could occur at such a site (EPA 1989b).

Scenario 2: Hypothetical Future Residential Exposure

Key variables in the potential future residential scenario, summarized in Tables 8-17 through 8-19, include soil ingestion rate (INGR), exposure frequency (EF), exposure duration (ED), soil to skin adherence factor (AF), skin surface area (SA), daily outdoor exposure time (ET), and inhalation rates (IR).

INGR of 200 mg/day and 100 mg/day for children aged 1 to 6 and all older receptor groups, respectively, were taken based on EPA guidance (EPA 1989c). EF for all receptor groups was taken as 365 days/year. ED was taken as 30 years corresponding to the upper 90th percentile time spent at one residence (EPA 1989b). AF was taken as 1.45 mg/cm^2 based upon the literature value reported by EPA (1989b) for potting soil. SA subject to contact with soil of 1,600 cm^2 for adults and teenagers was based upon assumed skin contact with the hands and one-third of the arms. SA for adolescents of 2,000 cm^2 was taken to correspond to the area of the hands and one-third of both arms and legs. SA for children was taken as the area of the hands and one-half of the arms and legs.

The selection of these skin areas was designed to correspond to the increased likelihood for skin parts to come into contact with soil as age decreases. ET was taken as 16 hour/day to correspond to an upper-bound daily annual average time spent in outdoor soil areas. Inhalation rates of 1.2 m^3/hr for adult males, females, and teenagers were based on EPA-recommended values for adults (EPA 1989b). Consistent with the methodology EPA used in deriving adult values, upperbound values for IR for adolescents and children of 1.5 m^3/hr and 1.2 m^3/hr, respectively, were derived based upon multiplying inhalation rates for light activities by a factor of 1.5 for a 10-year old and 6-year old (EPA 1989b).

Scenario 3: Residential Groundwater Usage

Key variables in the residential groundwater usage scenario, summarized in Tables 8-20 through 8-22, include water ingestion rate (INGR), exposure frequency (EF), exposure duration (ED), skin surface area (SA), exposure time while showering (ET), and inhalation rate (IR).

INGR of 2.0 L/day was based on the 90th percentile value recommended by EPA (1989b). EF was taken as 365 days per year. ED was taken as 30 years, the upper 90th percentile residence time (EPA 1989b). SA was taken as 1.8 m^2 based on the total body average skin area for adults (EPA 1989b). Showering ET, 0.2 hours, was based on the 90th percentile value reported by EPA (1989b). IR was taken as 0.6 m^3/hour, the mean value for adults engaged in light activity (1989b).

Methods for estimating the RMEs via all various complete exposure pathways associated with the Saunders site were described above. The exposure estimates are combined with toxicity estimates for each chemical as described in Section 8.4 to obtain risk estimates determined in Section 8.5. Also, the exposure estimates obtained by this process are given as chronic daily intakes (CDIs) or subchronic daily intakes (SDIs) for each complete pathway and exposure case in the risk estimation tables contained in Section 8.5.

8.3.9 Uncertainty in the Exposure Assessment

There are a number of factors that will cause the exposure levels estimated in this section to differ from the exposures that potential receptor populations may actually be experiencing. This section will

identify these factors, discuss the potential effects of the factors on the exposure estimates, and where possible and appropriate, estimate the degree of confidence that should be placed in the various assumptions and parameter estimates that have gone into the exposure estimates.

Environmental Sampling

Surface soil and groundwater samples collected during the RI were intended to characterize the nature and extent of contamination at the site. They were collected from the sampling locations in a systematic fashion and were selected to be representative of contaminant concentrations throughout the site soils, groundwater, surface water, or sediments. Upper 95% confidence limits or maximum concentration values were used as the source concentrations. This will tend to overestimate the concentrations in the source area as a whole.

Analytical Result Limitations

Two aspects of the analytical data marginally reduce the level of confidence in the estimates of contaminant concentrations in environmental media. One is the inclusion of estimated results (E and J flags) that may not have the same precision and accuracy as data meeting all of the standard QA criteria. This is a very minor concern.

The other aspect is the use of analytical detection limits that could allow potentially hazardous concentrations of some contaminants to go undetected. For example, the estimated cancer risk for dioxin in soil at the contract required quantitation limit (see Table 8-2) is 6×10^{-6}, which is above EPA's benchmark risk level of 10^{-6}. This source of uncertainty reduces the level of confidence that can be placed in the upper limit of the risk associated with environmental media in which these contaminants could be present at close to the detection limit. Although the inadequacy of analytical detection limits must not be overlooked, it is an uncertainty common to many risk assessments.

Exposure Estimation Calculations

The primary uncertainty regarding the exposure estimation calculations is that associated with the selection of appropriate parameter values. The values used and a brief rationale for their selection are

given in Section 8.3.8 (Tables 8-14 to 8-22), which describes the exposure calculations for the various pathways. Individual parameter values were selected so that the overall pathway exposure estimates would approximate the EPA definition of reasonable maximum exposure. It is important to note that in attempting to estimate the RME, it is generally not appropriate to use a worst-case or an upper 95th percentile estimate for every parameter in the calculation (EPA 1989a) as this will result in a pathway estimate that is so conservative it is in fact an extreme worst-case estimate (perhaps a 99.9999th percentile estimate), rather than a reasonable worst-case estimate, which would typically fall in the 99th to 99.9th percentile range.

Steady State Assumption

The exposure calculations used in this risk assessment all assume that the contaminant concentrations in the source media are at steady state and remain constant for the duration of the exposure periods. These range from a few years for future child residents to an entire 70-year composite lifetime for potential future residents assumed to be subject to direct contact with Saunders property soil. Since the waste disposal practices and conditions that resulted in the present contamination ceased or were changed at least 5 years ago, it is reasonable to assume that contaminant concentrations in the soil and groundwater are not increasing, and concentrations of metals that are persistent will remain constant whereas organics such as PCP and dioxin/furans may degrade and will probably decrease to some degree over the 6- to 70-year exposure periods of interest. Assuming that the source concentrations remain constant over those periods will probably overestimate to some degree the true exposures of organics.

Exposure Assessment Uncertainty Summary

Overall the exposure estimates obtained are probably moderately reliable. A number of the factors adding uncertainty to the estimates tend to result in overestimation of the exposure. These include:

- The use of upper 95% confidence limits, or the highest observed values, to estimate soil and groundwater exposure concentrations; and

- The use of the steady state assumption for source concentration estimates for PCP and dioxin/furans.

The cumulative effect of all of the exposure uncertainties most likely is to overestimate rather than underestimate the true potential exposures.

8.4 TOXICITY ASSESSMENT

The purpose of the toxicity assessment is to compile toxicity and carcinogenicity data for the chemicals of potential concern and to provide an estimate of the relationship between the extent of exposure to a contaminant and the likelihood and/or severity of adverse effects. The toxicity assessment will be accomplished in two steps--hazard identification and dose-response assessment.

Hazard identification is a qualitative description of the potential toxic properties of the chemicals of concern present on the Saunders Supply Company property. Brief toxicological profiles for the chemicals of concern are presented in Appendix K. These profiles present summaries of the potential toxic properties of contaminants of concern. In particular, they focus on a series of key toxicological endpoints and organ systems and will serve as the primary summaries of the potential hazards related to exposure to contaminants of concern.

The dose-response evaluation is a process that results in a quantitative estimate or index of toxicity for each contaminant at the site. For carcinogens, the index is the slope factor and for non-carcinogens, it is the reference dose. Section 8.4.1 categorizes practices and procedures used to develop quantitative indices of toxicity and to incorporate toxicological information into the risk estimation process. Section 8.4.2 discusses the relevance of ARARs, followed by Section 8.4.3, which discusses the limitations of the toxicity assessment.

8.4.1 Quantitative Indices of Toxicity

Quantitative indices of toxicity were compiled for the dose-response assessment to be used to estimate the relationship between the extent of exposure to a contaminant and the potential increased likelihood and/or severity of adverse effects. The methods for deriving

indices of toxicity and estimating potential adverse effects are presented below.

8.4.1.1 Categorization of Chemicals as Carcinogens or Noncarcinogens

For the purpose of this risk assessment, the chemicals of concern were divided into two groups: potential carcinogens and noncarcinogens. The risks posed by these two types of compounds are assessed differently because noncarcinogens generally exhibit a threshold dose below which no adverse effects occur, while it is typically assumed that no such threshold can be proven to exist for carcinogens.

As used here, the term carcinogen means any chemical for which there is sufficient evidence that exposure may result in continuing uncontrolled cell division (cancer) in humans and/or animals. Conversely, the term noncarcinogen means any chemical for which the carcinogenic evidence is negative or insufficient. These definitions are dynamic; compounds may be reclassified any time additional evidence becomes available that shifts the weight of evidence one way or the other.

Chemicals of concern have been classified as carcinogens or noncarcinogens based on weight-of-evidence criteria contained in the EPA Carcinogenicity Evaluation Guidelines (1986b). Table 8-23 summarizes the five EPA weight-of-evidence categories. According to these EPA guidelines, chemicals in the first two groups--A and B (B_1 or B_2)--are considered human carcinogens or probable human carcinogens based on sufficient evidence and should be the subject of nonthreshold carcinogenic risk estimation procedures. Depending upon the quality of the data, Group C chemicals may also be subjected to these procedures. The remaining chemicals--in Groups D and E--are defined as noncarcinogens and should be subjected to threshold-based toxicological risk estimation procedures.

8.4.1.2 Assessment of Noncarcinogens

Risks associated with noncarcinogenic effects (e.g., organ damage, immunological effects, birth defects, skin irritation) are usually assessed by comparing the estimated average exposure to the acceptable daily dose, now called the "reference dose" (RfD) by EPA. The RfD is

Table 8-23

FIVE EPA WEIGHT-OF-EVIDENCE
CATEGORIES FOR CHEMICAL CARCINOGENICITY

Group	Description
A	Human Carcinogen - sufficient evidence from epidemiological studies
B	Probable Human Carcinogen -
B1	o At least limited evidence of carcinogenicity to humans
B2	o Usually a combination of sufficient evidence for animals and inadequate data for humans
C	Possible Human Carcinogen - limited evidence of carcinogenicity in animals in the absence of human data
D	Not Classified - inadequate animal evidence of carcinogenicity
E	No Evidence of Carcinogenicity for Humans - no evidence of carcinogenicity in at least two adequate animal tests in different species or in both epidemiological and animal studies

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Source: EPA 1986b.

selected by identifying the lowest reliable not observed or lowest observed adverse effect level (NOAEL or LOAEL) in the scientific literature, then applying a suitable uncertainty factor (usually ranging from 10 to 1,000) to allow for differences between the study conditions and the human exposure situation to which the acceptable daily dose is to be applied. The five uncertainty factors commonly used are summarized in Table 8-24. NOAELs and LOAELs are usually based on laboratory experiments on animals in which relatively high doses are used. Consequently, uncertainty or safety factors are required when deriving RfDs to compensate for data limitations in the experiments and the lack of precision in extrapolating from high doses in animals to lower doses in humans.

RfDs are generally calculated using the formula:

$$\text{RfD (in mg/kg/day)} = \frac{\text{NOAEL or LOAEL (in mg/kg/day)}}{\text{Uncertainty Factor}}$$

If the estimated exposure exceeds the estimated acceptable intake, some adverse effects are presumed to be possible and that exposure level may be of potential concern. Conversely, if the estimated exposure is less than the estimated acceptable intake, no adverse effects would be expected and the exposure level is considered acceptable. Noncarcinogenic risks are usually assessed by calculating a hazard index, which is the ratio of the estimated exposure to the RfD as follows:

$$\text{HI} = \frac{\text{AVDI}}{\text{RfD}}$$

where

HI = Hazard Index

AvDI = Average Daily Intake (exposure)

RfD = Reference Dose (reference daily intake).

A hazard index greater than 1 indicates that adverse effects may be possible, while a value less than 1 means that adverse effects would not be expected.

Table 8-24

UNCERTAINTY FACTORS (MARGINS OF SAFETY) USED IN THE DERIVATION OF REFERENCE DOSES

Uncertainty Factor	Condition of Use
10	A 10-fold uncertainty factor is used with valid experimental results on appropriate durations of exposures of humans.
100	A 100-fold uncertainty factor is used when human data are not available and extrapolation is made from valid results of long-term animal studies.
1,000	A 1,000-fold uncertainty factor is used when human data are not available and extrapolation is made from animal studies of less than chronic exposure.
1-10	An additional uncertainty factor from 1 to 10 when using a lowest observed adverse effect level (LOAEL) instead of a no observed adverse effect level (NOAEL).
Intermediate uncertainty factor	Other uncertainty factors used, according to scientific judgment, when justified.

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Source: EPA 1986a.

EPA is in the process of developing subchronic RfDs based on potential noncarcinogenic effects associated with exposures ranging from a few weeks to seven years. Short-term exposures can occur when an activity resulting in exposure is performed for a limited period of time or when a chemical degrades or disperses to negligible concentrations within a short period. The hazard index for subchronic exposure is obtained by dividing the estimated average daily dose by the RfDs.

Chronic and subchronic RfDs for the oral exposure route are presented in Table 8-25. Chronic and subchronic RfDs for the inhalation exposure route are also presented in Table 8-25.

8.4.1.3 Assessment of Carcinogens

In contrast to noncarcinogenic effects, for which thresholds are thought to exist, scientists have been unable to demonstrate experimentally a threshold for carcinogenic effects. This has led to the assumption by federal regulatory agencies [e.g., EPA, Food and Drug Administration (FDA), and Occupational Safety and Health Administration (OSHA)] that any exposure to a carcinogen theoretically entails some finite risk of cancer. However, depending on the potency of a specific carcinogen and the level of exposure, such a risk could be vanishingly small.

Scientists have developed several mathematical models to estimate low-dose carcinogenic risks from observed high-dose risks. Consistent with current theories of carcinogenesis, EPA has selected the linearized multistage model based on prudent public health policy (EPA 1986b). In addition to using the linearized multistage model, EPA uses the upper 95% confidence limit for doses or concentrations in animal or human studies to estimate low-dose slope factors (SFs). By using these procedures, the regulatory agencies are unlikely to underestimate the actual slope factors (formerly called carcinogenic potency factors) for humans.

Using SFs, lifetime excess cancer risks can be estimated by:

$$\text{Risk} = \Sigma \text{LADI}_j \times \text{SF}_j$$

where

LADI_j = exposure route-specific lifetime average daily intake

SF_j = route-specific slope factor.

Table 8-25

CHRONIC AND SUBCHRONIC TOXICITY VALUES: POTENTIAL NONCARCINOGENIC EFFECTS

Chemical	RfD (mg/kg-day)	Confidence Level	Critical Effect	RfD Basis/ RfD Source	Uncertainty (UF) and Modifying (MF) Factors
ORAL ROUTE (CHRONIC)					
Arsenic	1×10^{-3}	NS	Cancer, keratosis	HEAST	UF = 1
Chromium III	1.0	Low	Hepatotoxicity	Diet/IRIS	UF = 100, MF = 10
Chromium VI	5×10^{-3}	Low	No effects reported	Water/IRIS	UF = 500, MF = 1
Copper	3.7×10^{-2}	NS	Local irritation	Water/HEAST	NA
Pentachlorophenol	3.0×10^{-2}	Medium	Liver and kidney pathology	Oral/IRIS	UF = 100, MF = 1
ORAL ROUTE (SUBCHRONIC)					
Arsenic	Same as Chronic	--	--	--	--
Chromium III	10	NS	Hepatotoxicty	Diet/HEAST	UF = 100
Chromium VI	2×10^{-2}	NS	No effects reported	Water/HEAST	UF = 100
Copper	Same as Chronic	NS	Local irritation	Water/HEAST	NA
Pentachlorophenol	Same as Chronic	--	Liver and kidney pathology	Oral/HEAST	UF = 100, MF = 1

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Key at end of table.

Table 8-25 (Cont.)

Chemical	RfD (mg/kg-day)	Confidence Level	Critical Effect	RfD Basis/ RfD Source	Uncertainty (UF) and Modifying (MF) Factors
INHALATION ROUTE (CHRONIC)					
Arsenic	1×10^{-3}	NS	Cancer, keratosis	Oral Chronic/HEAST	UF = 1
Chromium III	1.0	Low	Hepatotoxicity	Chronic Diet/IRIS	UF = 100, MF = 10
Chromium VI	5×10^{-3}	Low	No effects reported	Water Chronic/IRIS	UF = 500, MF = 1
Copper	3.7×10^{-2}	NS	Local irritation	Water Chronic/HEAST	NA
Pentachlorophenol	3×10^{-2}	Medium	Liver and kidney pathology	Oral Chronic/IRIS	UF = 100, MF = 1
INHALATION ROUTE (SUBCHRONIC)					
Arsenic	Same as Chronic	--	--	--	--
Chromium III	10	NS	Hepatotoxicty	Subchronic Diet/HEAST	UF = 100
Chromium VI	2×10^{-2}	NS	No effects reported	Subchronic Water/HEAST	UF = 100
Copper	Same as Chronic	--	--	--	--
Pentachlorophenol	Same as Chronic	--	--	--	--

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Key:

RfD = Reference dose
NA = Not available
NS = Not specified
IRIS = Integrated Risk Information System
HEAST = Health Effects Assessment Summary Tables

Compiled by Ecology and Environment, Inc., 1991.

Using the multistage model, the carcinogenic risks for the oral, dermal, and inhalation routes of exposure are calculated as follows:

$$\text{Risk} = LADI_o SF_o + LADI_d SF_o + LADI_i SF_i$$

where subscript "o" indicates the oral route, subscript "d" the dermal route, and subscript "i" the inhalation route. SFs for the chemicals of concern for the oral and inhalation exposure routes are presented in Table 8-26. EPA's weight-of-evidence classification for the chemical and the type of cancer that may be associated with exposure to the chemical are included.

There have been several suggestions recently that EPA's current cancer potency estimate (slope factor) for dioxin is too high and results in overestimation of the risks dioxin exposure may pose. In response, EPA has agreed to reevaluate all available information on dioxin toxicity in order to either verify its present toxicity estimate or adjust it as required. If a change is made in EPA's cancer potency estimate for dioxin, the estimated risks due to potential dioxin exposure at the Saunders site also would change proportionately.

Once substances have been absorbed by the oral or dermal routes, their distribution, metabolism, and elimination patterns (pharmacokinetics) are usually similar. For this reason, and because dermal route RfDs and SFs are usually not available, oral route RfDs and SFs are commonly used to evaluate exposures to substances by both the oral and dermal routes. This approach is not appropriate and is not used if the adverse effect occurs at the point of exposure. Examples would be skin irritation or skin cancer resulting from dermal exposure. Although inhalation route pharmacokinetics differ more from oral route kinetics than do the dermal route kinetics, oral RfDs and SFs may also be used to evaluate inhalation exposures (except in the case of exposure point effects) if inhalation route RfDs and SFs are not available.

Exposure to some chemicals may result in both carcinogenic and noncarcinogenic effects. In these cases, both the carcinogenic and noncarcinogenic effects were evaluated and considered in the risk assessment process.

Table 8-26

TOXICITY VALUES: POTENTIAL CARCINOGENIC EFFECTS-
CONFIDENCE IN CLASSIFICATION AND IN SLOPE FACTOR (SF)

Chemical	Slope Factor (SF) $(mg/kg-day)^{-1}$	Weight-of-Evidence Classification	Type of Cancer	SF Basis/SF Source
ORAL ROUTE				
Arsenic	1.75(g)	A	Skin	Drinking Water Humans/HEAST
Pentachlorophenol	0.12	B2	--	Diet/HEAST
TCDD	1.5×10^5	B2	--	Diet/IRIS
INHALATION ROUTE				
Arsenic	50	A	Respiratory Tract	Occupational Air/IRIS
Chromium (VI)	4.1	A	Lung	Occupational Air/IRIS
Pentachlorophenol	0.12	B2	--	Diet/IRIS
TCDD	1.5×10^5	B2	--	Diet/IRIS

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Key:

-- = Not specified
IRIS = Integrated Risk Information System
HEAST = Health Effects Assessment Summary Tables
(g) = Proposed by Risk Assessment Forum and scheduled for Science Advisory Board Review

Compiled by Ecology and Environment, Inc., 1991.

Since frequently no "safe" exposure can be demonstrated to exist for carcinogens, the risk management task becomes one of determining what level of risk will be deemed acceptable. In general, regulatory agencies in the United States (EPA, FDA, and OSHA) have not established a uniform cancer risk level for distinguishing between risks that are deemed acceptable and those that may be of concern. The agencies have generally considered risks in the range of one in 10,000 (1×10^{-4}) to one in 10,000,000 (1×10^{-7}) to be acceptable. EPA has recently adopted the policy that acceptable exposures are generally those that represent an excess upper bound lifetime cancer risk to an individual of between 10^{-4} and 10^{-6}. In addition, EPA will use the 10^{-6} risk level as the point of departure for determining remediation goals for National Priorities List sites (EPA 1990).

8.4.2 Uncertainties Related to the Toxicity Assessment

In order to evaluate the meaning of any risk assessment, one must consider the uncertainties in the assumptions made, the impact of changing the magnitude of those assumptions on the risk estimates, and the relevance of the findings to real world exposures and risks. Due to the number of assumptions, data points, and calculations, a degree of uncertainty is necessarily associated with the numerical toxicity values in any risk assessment.

This section begins with a discussion of the assumptions used to estimate noncarcinogenic risks, continues with a discussion of carcinogenic risk estimates, and concludes with a discussion of the other major assumptions used in developing the exposure scenarios.

8.4.2.1 Evaluation of Noncarcinogenic Toxicity Assessment Assumptions

Key assumptions used in assessing the likelihood of noncarcinogenic effects are that threshold doses exist below which various noncarcinogenic effects do not occur and that the occurrence or absence of noncarcinogenic effects can be extrapolated between species and occasionally between routes of exposure and over varying exposure durations. The threshold assumption appears to be sound for most noncarcinogens based on reasonably good fits of experimental data to the usual dose response curves. One possible exception to this is lead, which may not have a

threshold base for its noncarcinogenic effects (EPA 1988b). However, lead was not a compound of concern at the Saunders facility.

The other assumption generally appears to be true to varying degrees. The effects observed in one species or by one route of exposure may not occur in another species or by another route, or they may occur at a higher or lower dose due to differences in the pharmacokinetics (uptake, distribution, metabolism, and elimination) of a compound in different species or by different routes of exposure. The uncertainty in these assumptions is taken into account in the development of RfDs through the use of safety or uncertainty factors. The uncertainty factors used by EPA are conservative (health protective) in nature in that they tend to overestimate the uncertainties so that the RfDs obtained are unlikely to be too high. Use of the resulting RfDs tends to overestimate the potential for noncarcinogenic effects occurring at a given exposure level. Section 8.4.4 discussed the uncertainty factors used to derive the RfDs for chemicals of concern at the Saunders site.

Uncertainty factors used to derive RfDs are presented in Table 8-25 for each chemical of concern. For example, an uncertainty factor of 100 was used to derive the RfD for trivalent chromium: 10 for species-to-species extrapolation and 10 to protect sensitive individuals. In addition to uncertainty factors, a modifying factor is applied to reflect a qualitative professional assessment of additional uncertainties in the critical study and in the entire data base for the chemical not explicitly addressed by the preceding uncertainty factors. The modifying factor ranges from >0 to 10 with a default value of 1 (EPA 1989a).

For example, confidence in the oral RfD for trivalent chromium as defined in IRIS is low because of lack of explicit detail on study protocol and results and reflects the lack of high-dose supporting data. The low confidence reflects the foregoing, but also reflects the lack of an observed effect level. Low confidence in the RfD follows. Confidence levels for verified RfDs are included in Table 8-25.

8.4.2.2 Evaluation of Carcinogenic Toxicity Assessment Assumptions

The chemicals of concern have been evaluated by EPA using its weight-of-evidence carcinogenicity evaluation criteria and have been

placed in Group A, human carcinogens, or Group B, probable human carcinogens, based on sufficient data in humans and animals and insufficient data in humans, respectively (EPA 1986b).

Rodent bioassay and epidemiological studies, such as those performed for the chemicals of concern, would require tens of thousands of animals or humans in order to determine whether or not a chemical may be carcinogenic at low doses. As the relationship between tumor location, time to appearance, and the proportion of animals with cancer determines the estimated carcinogenic SF, animal bioassay or human epidemiological data are not routinely sufficient for directly estimating SF at low doses. Therefore, by necessity, agencies such as EPA use carcinogenic extrapolation models for estimating low dose SFs. Based on policy grounds, these agencies assume that there is no threshold dose below which carcinogenic risks will not occur. This is equivalent to the assumption that every dose above zero, no matter how low, carries with it a small but finite risk of cancer. The agencies also assume that the dose-response relationship is linear at low doses. This is contrary to approaches used for other toxic effects, because thresholds are assumed to exist.

The current model favored by EPA and certain other federal regulatory agencies is the linearized multistage model. The agency then uses the statistically derived upper 95% confidence bounds, rather than a maximum likelihood value for SF. The agency has concluded, based on theoretical grounds consistent with human epidemiological and animal data, that cancer follows a series of discrete stages (i.e., initiation, promotion, and progression) that ultimately can result in the uncontrolled cell proliferation known as cancer. Consistent with this conclusion, the use of the linearized multistage model permits an estimation of SF that is not likely to be exceeded if the real slope could be measured. However, compelling scientific arguments can be made for several other extrapolative models, which, if used, could result in significantly reduced values for SFs, some tens of millions of times lower than those estimated using the linearized multistage model. The one hit model used to estimate risks due to exposures above the linear range of the multistage model is one such model. Thus, the current EPA SFs calculated in this fashion represent upper-bound values based on animal

data that should not be interpreted as necessarily equivalent to actual human cancer potencies. It is this conservative value, nevertheless, that is used in this risk assessment on policy grounds for the protection of public health.

8.4.2.3 Summary of Toxicity Assessment Uncertainties

The basic uncertainties underlying the assessment of the toxicity of a chemical include:

- Uncertainties arising from the design, execution, or relevance of the scientific studies that form the basis of the assessment; and
- Uncertainties involved in extrapolating from the underlying scientific studies to the exposure situation being evaluated, including variable responses to chemical exposures within human and animal populations, between species, and between routes of exposure.

These basic uncertainties could result in a toxicity estimate, based directly on the underlying studies, that either under- or overestimates the true toxicity of a chemical in the circumstances of interest.

The toxicity assessment process compensates for these basic uncertainties through the use of safety factors (uncertainty factors) and modifying factors, when assessing noncarcinogens, and the use of the upper 95% confidence limit from the linearized multistage model for the SF when assessing carcinogens. The use of the safety factors and the upper 95% confidence limit in deriving the RfDs and SFs ensures that the toxicity values used in the risk estimation process are very unlikely to underestimate, and thus, almost always overestimate, the true toxicity of a chemical.

8.5 RISK CHARACTERIZATION

This section combines the information developed in the exposure and toxicity assessment sections to obtain estimates of the risks posed by the Saunders facility contaminants to human health. The process by which this is done is as follows:

Risks due to carcinogenic and noncarcinogenic contaminants are assessed differently, as discussed in Section 8.4.1. Briefly,

carcinogenic risks are assessed by multiplying the estimated chronic daily intake (CDI) of a carcinogen by its estimated slope factor (SF) to obtain the estimated risk, expressed as the probability of that exposure resulting in an excess incidence of cancer.

The potential for adverse effects resulting from exposure to noncarcinogens is assessed by comparing the CDI or subchronic daily intake (SDI) of a substance to its chronic or subchronic RfD. This comparison is performed by calculating the ratio of the estimated CDI or SDI to the corresponding RfD, which is called a hazard quotient or hazard index. If the hazard index is less than 1, no adverse effects would be expected; however, if it is greater than 1, adverse effects could be possible.

The excess cancer risk or the hazard quotient for exposure to each chemical by each route of exposure, exposure pathway, category of receptor (i.e., adult or child), and exposure case (RME) are initially estimated separately.

The separate cancer risk estimates are then summed across chemicals and across all exposure routes and pathways applicable to the same population to obtain the total excess cancer risk for that population. Hazard quotients for noncarcinogens are summed across chemicals that produce the same type of adverse effect (such as liver damage) but are kept separate if their effects are different. Hazard quotients for subchronic and chronic effects are separately summed across all chemicals, exposure routes, and pathways applicable to the same population to obtain hazard indices for that population. Finally, risks that could potentially occur under current land use conditions are summed separately from those that could only occur if future land use differs from its current use.

Section 8.5.1 presents a number of tables that contain the detailed risk estimates just described. Section 8.5.2 discusses uncertainties associated with the risk estimates. Section 8.5.3 summarizes the risk estimation results and identifies the chemicals, pathways, and receptors that account for the most significant risks at the Saunders site.

8.5.1 Risk Estimates

This section presents a number of tables (8-27 through 8-36) that contain exposure and toxicity estimates, along with key items of qualifying or supporting information carried forward from the exposure and toxicity assessment sections of this report. The tables also contain the excess cancer risk estimates and the hazard quotients obtained by combining the exposure and toxicity information as described above. Descriptions of the numerical entries contained in these tables are presented below.

Entries Pertaining to Exposure Estimates

The exposure estimates are given as either CDIs or SDIs as befits the chemical and pathway under consideration. The next column indicates whether the CDI or SDI has been adjusted for the absorption efficiency of the exposure route. Generally, the original literature data that forms the basis for EPA's cancer potency SFs and RfDs report administered doses rather than absorbed doses; therefore, the SFs and RfDs are usually derived on an administered dose basis as well. It is important that the exposure estimate be expressed on the same basis as the corresponding SF or RfD. Thus, it is usually not appropriate to adjust oral or inhalation route exposure estimates for absorption. An exception would be, for example, if an oral SF or RfD was being used in lieu of an inhalation SF or RfD that was unavailable and the relationship between the chemical's absorption by those routes was known. Dermal exposure estimates usually are adjusted for adsorption if the adverse effect appears to result from systemic exposure (e.g., liver, kidney or neurological effects) but not if the effect is a dermal lesion (e.g., skin irritation or skin cancer) that occurs at the point of exposure.

Entries Pertaining to Carcinogens

SF is the chemical's estimated cancer potency slope factor, a quantitative estimate of a chemical's ability to cause cancer. The weight of evidence indicates the type and strength (weight) of the scientific information upon which a chemical's classification as a carcinogen is based. The formal definitions of the weight of evidence categories were

given in Section 8.4.2 and presented in Table 8-23. Briefly, they are as follows: Group A chemicals are considered confirmed human carcinogens; Group B1 and B2 chemicals, probable human carcinogens; and Group C chemicals, possible human carcinogens. Group D indicates that there is insufficient evidence to classify the chemical as a carcinogen and Group E chemicals are confirmed noncarcinogens. Group A, B, and C chemicals are evaluated as carcinogens while Group D and E chemicals are evaluated as noncarcinogens. The type of cancer caused by a chemical is identified in the tables only for Group A carcinogens.

The SF source is the source or reference for the SF value used. The preferred source is EPA's integrated risk information system (IRIS) data base, which contains confirmed values reflecting the consensus judgment of the agency. The second choice is the EPA's health effects assessment summary tables (HEAST), which contain information taken from final documents prepared by the EPA Office of Health and Environmental Assessment. The third choice are values from other EPA documents, and the fourth choice would be values derived directly from the general literature.

The SF basis is the vehicle in which the chemical was administered or the medium of exposure. The chemical specific risk, total pathway risk, and total exposure risk are the initially calculated risk and the summation of risks over chemicals, exposure routes, and exposure pathways.

Entries Pertaining to Noncarcinogens

The RfD is the chronic or subchronic reference dose, the dosage below which no adverse effects are expected. The confidence level indicates the degree of confidence that should be placed in the RfD value and is usually obtained from the IRIS entry for a chemical. The critical effect is the effect or target organ affected by the smallest dose of the chemical that produces any adverse effect and that serves as the basis for the RfD. The RfD source is the source or reference for the RfD. The RfD source should be selected in the same hierarchical fashion as the SF discussed above. The RfD basis is the vehicle in which the chemical was administered or the medium of exposure in the study(ies) that served as the basis for the RfD. RfD uncertainty

adjustments indicate what adjustments have been made in deriving the RfD value to allow for uncertainties arising from variation in human sensitivity, animal to human, route to route, or LOAEL to NOAEL extrapolations. The modifying factor is an additional adjustment factor based on professional judgment used to compensate for factors other than the usual uncertainty adjustments. The hazard quotient, pathway hazard index, and total exposure hazard index are the initially calculated CDI/RfD or SDI/RfD ratios and the summation of like ratios over chemicals, exposure routes, and exposure pathways.

Risk Estimation Summary Tables

Tables 8-27 and 8-28 present cancer and noncancer risk estimates, respectively, for the current use worker scenario (Scenario 1). For this scenario the total cancer risk for all exposure pathways exceeds the 10^{-6} risk level, whereas the hazard index is less than one.

Tables 8-29 and 8-30 present cancer risk estimates for an adult over an entire (composite) lifetime and for the entire period of exposure for a 1- to 6-year-old child (Scenario 2). For both exposure periods the total cancer risk exceeds the 10^{-6} risk level. Tables 8-31 and 8-32 present the corresponding noncancer hazard indices for these two receptor groups. The hazard index is less than one for both groups.

Tables 8-33 and 8-34 present cancer risk estimates for adult males for residential groundwater use of the lower and upper aquifers, respectively (Scenario 3). Both cancer risk estimates exceed the 10^{-6} risk level. Tables 8-35 and 8-36 present the corresponding noncancer hazard indices. The hazard index for the lower aquifer is less than one. The hazard index for the upper aquifer is 12.

8.5.2 Risk Characterization Uncertainties

The risk characterization combines and integrates the information developed in the exposure and toxicity assessments; therefore, uncertainties associated with these assessments also affect the degree of confidence that can be placed in risk characterization results. The reader is referred to Sections 8.3.9 and 8.4.3 for full discussions of the factors causing uncertainty in the exposure and toxicity assessments, respectively. The primary factors contributing to exposure and toxicity uncertainties are briefly reviewed here.

Table 8-27

CANCER RISK ESTIMATES
CURRENT USE
WORKER SOIL EXPOSURES
REASONABLE MAXIMUM EXPOSURE - ADULT

Chemical	CDI (mg/kg-day)	CDI Adj. for Absorp.	SF (mg/kg-day)$^{-1}$	Weight of Evidence	Type of Cancer	SF Source	SF Basis (Vehicle)	Chemical-Specific Risk	Total Pathway Risk	Total Exposure Risk
Exposure Pathway:	**Ingestion of Soil, Adult**									
Arsenic	6.0×10^{-5}	No	1.75	A	Skin	HEAST	Drinking Water	1.0×10^{-4}	--	--
Pentachlorophenol	9.1×10^{-7}	No	0.12	B2	--	HEAST	Diet	1.1×10^{-7}	--	--
2,3,7,8-TCDD Equivalents	1.1×10^{-9}	No	1.5×10^{5}	B2	--	IRIS	Diet	1.6×10^{-4}	--	--
									2.6×10^{-4}	--
Exposure Pathway:	**Dermal Contact with Soil, Adult**									
Arsenic	6.9×10^{-6}	Yes	1.75	A	Skin	HEAST	Drinking Water	1.2×10^{-5}	--	--
Pentachlorophenol	1.0×10^{-6}	Yes	0.12	B2	--	HEAST	Diet	1.3×10^{-7}	--	--
2,3,7,8-TCDD Equivalents	1.2×10^{-10}	Yes	1.5×10^{5}	B2	--	IRIS	Diet	1.7×10^{-5}	--	--
									3.1×10^{-5}	

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Key at end of table.

Table 8-27 (Cont.)

Chemical	CDI (mg/kg-day)	CDI Adj. for Absorp.	SF (mg/kg-day)$^{-1}$	Weight of Evidence	Type of Cancer	SF Source	SF Basis (Vehicle)	Chemical-Specific Risk	Total Pathway Risk	Total Exposure Risk
Exposure Pathway: Inhalation of Soil Particulate, Adult										
Arsenic	1.2×10^{-6}	No	50	A	Respiratory Tract	IRIS	Occ. Air	6.0×10^{-5}	--	--
Chromium (VI)	5.1×10^{-9}	No	41	A	Lung	IRIS	Occ. Air	2.1×10^{-7}	--	--
Pentachlorophenol	1.8×10^{-8}	No	0.12	B2	--	HEAST	Diet	2.1×10^{-9}	--	--
2,3,7,8-TCDD Equivalents	2.1×10^{-11}	No	1.5×10^{5}	B2	--	IRIS	Diet	3.2×10^{-6}	--	--
									6.3×10^{-5}	
Adult Male--worker soil exposures--Total Cancer Risk (Weight of evidence predominantly A)										3.6×10^{-4}*

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Key:

* *Does not* total exactly due to independent rounding of total pathway risks.
-- = Not specified.
IRIS = Integrated Risk Information System.
HEAST = Health Effects Assessment Summary Tables.

Source: Ecology and Environment, Inc. 1991.

Table 8-28

CHRONIC HAZARD INDEX ESTIMATES
CURRENT USE
WORKER SOIL EXPOSURES
REASONABLE MAXIMUM EXPOSURE - ADULT

Chemical	CDI (mg/kg-day)	CDI Adj. for Absorp.	RfD (mg/kg-day)	Confidence Level	Critical Effect	RfD Source	RfD Basis	RfD Uncertainty Adj.	Modif. Factor	Hazard Quotient	Pathway Hazard Index	Total Exposure Hazard Index
Exposure Pathway: Ingestion of Soil												
Arsenic	1.0×10^{-4}	No	0.001	--	Cancer, Keratosis	HEAST	Oral	1	--	0.10	--	--
Chromium (Tot., III)	1.2×10^{-4}	No	1.0	Low	Hepatotoxicity	IRIS	Diet	100	10	1.2×10^{-4}	--	--
Chromium (VI)	4.5×10^{-7}	No	0.005	Low	No effect	IRIS	Water	500	1	9.0×10^{-5}	--	--
Copper	8.8×10^{-5}	No	0.037	--	Local Irritation	HEAST	Water	--	--	2.3×10^{-3}	--	--
Pentachlorophenol	1.6×10^{-6}	No	0.03	Medium	Liver and Kidney Pathology	IRIS	Oral	100	1	5.2×10^{-5}	--	--
											0.10	

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Key at end of table.

Table 8-28 (Cont.)

Chemical	CDI (mg/kg·day)	CDI Adj. for Absorp.	RfD (mg/kg·day)	Confidence Level	Critical Effect	RfD Source	RfD Basis	RfD Uncertainty Adj.	Modif. Factor	Hazard Quotient	Pathway Hazard Index	Total Exposure Hazard Index
Exposure Pathway: Dermal Contact with Soil												
Arsenic	1.2×10^{-5}	Yes	0.001	--	Cancer, Keratosis	HEAST	Oral	1	--	1.2×10^{-2}	--	--
Chromium (Tot., III)	1.3×10^{-5}	Yes	1.0	Low	Hepatotoxicity	IRIS	Diet	100	10	1.3×10^{-5}	--	--
Chromium (VI)	5.2×10^{-8}	Yes	0.005	Low	No effect	IRIS	Water	500	1	1.0×10^{-5}	--	--
Copper	9.9×10^{-6}	Yes	0.037	--	Local Irritation	HEAST	Water	--	--	2.7×10^{-4}	--	--
Pentachlorophenol	1.8×10^{-6}	Yes	0.03	Medium	Liver and Kidney Pathology	IRIS	Oral	100	1	6.0×10^{-5}	--	--
											0.01	

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Key at end of table.

Table 8-28 (Cont.)

Chemical	CDI (mg/kg·day)	CDI Adj. for Absorp.	RfD (mg/kg·day)	Confidence Level	Critical Effect	RfD Source	RfD Basis	RfD Uncertainty Adj.	Modif. Factor	Hazard Quotient	Pathway Hazard Index	Total Exposure Hazard Index
Exposure Pathway: Inhalation of Soil Particulate												
Arsenic	2.1×10^{-6}	No	0.001	--	Cancer, Keratosis	HEAST	Oral	1	--	2.1×10^{-3}	--	--
Chromium (Tot., III)	2.3×10^{-6}	No	1.0	Low	Hepatotoxicity	IRIS	Diet	100	10	2.3×10^{-6}	--	--
Chromium (VI)	9.0×10^{-9}	No	0.005	Low	No effect	IRIS	Water	500	1	1.8×10^{-6}	--	--
Copper	1.7×10^{-6}	No	0.037	--	Local Irritation	HEAST	Water	--	--	4.6×10^{-5}	--	--
Pentachlorophenol	3.1×10^{-8}	No	0.03	Medium	Liver and Kidney Pathology	IRIS	Oral	100	1	1.0×10^{-6}	--	--
											2.1×10^{-3}	
Adult Male -- worker exposures -- Total Chronic Hazard Index												0.12*

02[UZ]ZD3081:D3123/4101/1

Key:

-- = Not specified.
IRIS = Integrated Risk Information System.
HEAST = Health Effects Assessment Summary tables.

*Does not total exactly due to independent rounding of pathway total hazard indices.

Source: Ecology and Environment, Inc. 1991.

Table 8-29

CANCER RISK ESTIMATES
FUTURE USE
RESIDENTIAL SOIL EXPOSURES
REASONABLE MAXIMUM EXPOSURE - COMPOSITE LIFETIME

Chemical	CDI (mg/kg-day)	CDI Adj. for Absorp.	SF $(mg/kg-day)^{-1}$	Weight of Evidence	Type of Cancer	SF Source	SF Basis (Vehicle)	Chemical-Specific Risk	Total Pathway Risk	Total Exposure Risk
Exposure Pathway: *Ingestion of Soil, Adult*										
Arsenic	3.1×10^{-5}	No	1.75	A	Skin	HEAST	Drinking Water	5.4×10^{-5}	--	--
Pentachlorophenol	1.2×10^{-4}	No	0.12	B2	--	HEAST	Diet	1.5×10^{-5}	--	--
2,3,7,8-TCDD Equivalents	4.7×10^{-9}	No	1.5×10^{5}	B2	--	IRIS	Diet	7.0×10^{-4}	--	--
									7.7×10^{-4}	
Exposure Pathway: Dermal Contact with Soil, Adult										
Arsenic	6.0×10^{-6}	Yes	1.75	A	Skin	HEAST	Drinking Water	1.1×10^{-5}	--	--
Pentachlorophenol	2.4×10^{-4}	Yes	0.12	B2	--	HEAST	Diet	2.8×10^{-5}	--	--
2,3,7,8-TCDD Equivalents	9.0×10^{-10}	Yes	1.5×10^{5}	B2	--	IRIS	Diet	1.4×10^{-4}	--	--
									1.8×10^{-4}	

02[UZ]ZD3081:D3123/4105/2

Key at end of table.

Table 8-29 (Cont.)

Chemical	CDI (mg/kg·day)	CDI Adj. for Absorp.	SF $(mg/kg_i \cdot day)^{-1}$	Weight of Evidence	Type of Cancer	SF Source	SF Basis (Vehicle)	Chemical-Specific Risk	Total Pathway Risk	Total Exposure Risk
Exposure Pathway: Inhalation of Soil Particulate, Adult										
Arsenic	4.8×10^{-7}	No	50	A	Respiratory Tract	IRIS	Occ. Air	2.4×10^{-5}	--	--
Chromium (VI)	9.6×10^{-9}	No	41	A	Lung	IRIS	Occ. Air	3.9×10^{-7}	--	--
Pentachlorophenol	1.9×10^{-6}	No	0.12	B2	--	HEAST	Diet	2.2×10^{-7}	--	--
2,3,7,8-TCDD Equivalents	7.3×10^{-11}	No	1.5×10^{5}	B2	--	IRIS	Diet	1.1×10^{-5}	3.6×10^{-5}	--
Adult residential soil exposures--Total Cancer Risk (weight of evidence predominantly A)										9.9×10^{-4}*

02[UZ]ZD3081:D3123/4105/2

Key:

-- = Not specified.
IRIS = Integrated Risk Information System.
HEAST = Health Effects Assessment Summary Tables.

*Does not total exactly due to independent rounding of chemical-specific and total pathway risks.

Source: Ecology and Environment, Inc. 1991.

Table 8-30

CANCER RISK ESTIMATES
FUTURE USE
RESIDENTIAL SOIL EXPOSURES
REASONABLE MAXIMUM EXPOSURE - CHILD (1 to 6 YEAR OLD)

Chemical	CDI (mg/kg-day)	CDI Adj. for Absorp.	SF ($(mg/kg-day)^{-1}$)	Weight of Evidence	Type of Cancer	SF Source	SF Basis (Vehicle)	Chemical-Specific Risk	Total Pathway Risk	Total Exposure Risk
Exposure Pathway:	**Ingestion of Soil, Child**									
Arsenic	1.4×10^{-5}	No	1.75	A	Skin	HEAST	Drinking Water	2.5×10^{-5}	--	--
Pentachlorophenol	5.6×10^{-5}	No	0.12	B2	--	HEAST	Diet	6.7×10^{-6}	--	--
2,3,7,8-TCDD Equivalents	2.1×10^{-9}	No	1.5×10^{5}	B2	--	IRIS	Diet	3.2×10^{-4}	--	--
									3.5×10^{-4}*	
Exposure Pathway:	**Dermal Contact with Soil, Child**									
Arsenic	1.9×10^{-6}	Yes	1.75	A	Skin	HEAST	Drinking Water	3.3×10^{-6}	--	--
Pentachlorophenol	7.2×10^{-5}	Yes	0.115	B	--	EPA (1986)	Oral	8.8×10^{-6}	--	--
2,3,7,8-TCDD Equivalents	2.8×10^{-10}	Yes	1.5×10^{5}	B2	--	IRIS	Diet	4.2×10^{-5}	--	--
									5.4×10^{-5}*	

02[UZ]ZD3081:D3123/4106/3

Key at end of table.

Table 8-30 (Cont.)

Chemical	CDI (mg/kg-day)	CDI Adj. for Absorp.	SF ($mg/kg-day)^{-1}$	Weight of Evidence	Type of Cancer	SF Source	SF Basis (Vehicle)	Chemical-Specific Risk	Total Pathway Risk	Total Exposure Risk
Exposure Pathway: Inhalation of Soil Particulate, Child										
Arsenic	1.4×10^{-7}	No	50	A	Respiratory Tract	IRIS	Occ. Air	6.9×10^{-6}	--	--
Chromium (VI)	2.7×10^{-9}	No	41	A	Lung	IRIS	Occ. Air	1.1×10^{-7}	--	--
Pentachlorophenol	5.3×10^{-7}	No	0.12	B2	--	HEAST	Diet	6.4×10^{-8}	--	--
2,3,7,8-TCDD Equivalents	2.1×10^{-11}	No	1.5×10^{5}	B2	--	IRIS	Diet	3.1×10^{-6}	--	--
									1.0×10^{-5}	
Child--residential soil exposures--Total Cancer Risk (weight of evidence predominantly A)										4.0×10^{-4}*

02[UZ]ZD3081:D3123/4106/3

Key:

-- = Not specified.
* = Does not total exactly due to independent rounding of chemical-specific and total pathway risks.
IRS = Integrated Risk Information System.
HEAST = Health Effects Assessment Summary Tables.

Source: Ecology and Environment, Inc. 1991.

Table 8-31

CHRONIC HAZARD INDEX ESTIMATES
FUTURE USE
RESIDENTIAL SOIL EXPOSURES
REASONABLE MAXIMUM EXPOSURE - ADULT

Chemical	CDI (mg/kg·day)	CDI Adj. for Absorp.	RfD (mg/kg·day)	Confidence Level	Critical Effect	RfD Source	RfD Basis	RfD Uncertainty Adj.	Modif. Factor	Hazard Quotient	Pathway Hazard Index	Total Exposure Hazard Index
Exposure Pathway:	**Ingestion of Soil**											
Arsenic	$2.3x10^{-5}$	No	0.001	--	Cancer, Keratosis	HEAST	Oral	1	--	$2.3x10^{-2}$	--	--
Chromium (Total, III)	$3.6x10^{-5}$	No	1.0	Low	Hepatotoxicity	IRIS	Diet	100	10	$3.6x10^{-5}$	--	--
Chromium (VI)	$6.7x10^{-7}$	No	0.005	Low	No effect	IRIS	Water	500	1	$9.1x10^{-5}$	--	--
Copper	$2.6x10^{-5}$	No	0.037	--	Local Irritation	HEAST	Water	--	--	$7.0x10^{-4}$	--	--
Pentachlorophenol	$8.9x10^{-5}$	No	0.03	Medium	Liver and Kidney Pathology	IRIS	Oral	100	1	$3.0x10^{-3}$	--	--
											$2.7x10^{-2}$*	
Exposure Pathway:	**Dermal Contact with Soil**											
Arsenic	$5.4x10^{-6}$	Yes	0.001	--	Cancer, Keratosis	HEAST	Oral	1	--	$5.3x10^{-3}$	--	--
Chromium (Total, III)	$8.2x10^{-6}$	Yes	1.0	Low	Hepatotoxicity	IRIS	Oral	100	10	$8.2x10^{-6}$	--	--
Chromium (VI)	$1.1x10^{-7}$	Yes	0.005	Low	No effect	IRIS	Inhalation	500	1	2.1x10-5	--	--

02[UZ]ZD3081:D3123/4107/2

Key at end of table.

Table 8-31 (Cont.)

Chemical	CDI (mg/kg-day)	CDI Adj. for Absorp.	RfD (mg/kg-day)	Confidence Level	Critical Effect	RfD Source	RfD Basis	RfD Uncertainty Adj.	Modif. Factor	Hazard Quotient	Pathway Hazard Index	Total Exposure Hazard Index
Copper	6.0×10^{-6}	Yes	0.037	--	Local Irritation	HEAST	Diet	--	--	1.6×10^{-4}	--	--
Pentachlorophenol	2.1×10^{-4}	Yes	0.03	Medium	Liver and Kidney Pathology	IRIS	Inhalation	100	1	6.9×10^{-3}	--	--
											0.01*	
Exposure Pathway: Inhalation of Soil Particulate												
Arsenic	4.4×10^{-7}	No	0.001	--	Cancer, Keratosis	HEAST	Oral	1	--	4.4×10^{-4}	--	--
Chromium (Total, III)	6.8×10^{-7}	No	1.0	Low	Hepatotoxocity	IRIS	Diet	100	10	6.9×10^{-7}	--	--
Chromium (VI)	8.8×10^{-9}	No	0.005	Low	No effect	IRIS	Water	500	1	1.8×10^{-6}	--	--
Copper	5.0×10^{-7}	No	0.037	--	Local Irritation	HEAST	Water	--	--	1.3×10^{-5}	--	--
Pentachlorophenol	1.7×10^{-6}	No	0.03	Medium	Liver and Kidney Pathology	IRIS	Oral	100	1	5.7×10^{-5}	--	--
											5.1×10^{-4}*	
Adult Residential soil exposures -- Total Chronic Hazard Index												4.0×10^{-2}*

02[UZ]ZD3081:D3123/4107/2

Key:

-- = Not specified.
* = Does not total exactly due to independent rounding.
IRIS = Integrated Risk Information System.
HEAST = Health Effects Assessment Summary Tables.

Source: Ecology and Environment, Inc. 1991.

Table 8-32

SUBCHRONIC HAZARD INDEX ESTIMATES
FUTURE USE
RESIDENTIAL SOIL EXPOSURES
REASONABLE MAXIMUM EXPOSURE - CHILD

Chemical	CDI (mg/kg-day)	CDI Adj. for Absorp.	RfD (mg/kg-day)	Confidence Level	Critical Effect	RfD Source	RfD Basis	RfD Uncertainty Adj.	Modif. Factor	Hazard Quotient	Pathway Hazard Index	Total Exposure Hazard Index
Exposure Pathway:	**Ingestion of Soil**											
Arsenic	2.0×10^{-4}	No	0.001	--	Cancer, Keratosis	HEAST	Oral	1	--	2.0×10^{-1}	--	--
Chromium (Total, III)	3.1×10^{-4}	No	10	--	Hepatotoxicity	HEAST	Diet	100	--	3.1×10^{-5}	--	--
Chromium (VI)	4.0×10^{-6}	No	0.02	--	No effect	HEAST	Water	100	1	2.0×10^{-4}	--	--
Copper	2.3×10^{-4}	No	0.037	--	Local Irritation	HEAST	Water	--	--	6.1×10^{-3}	--	--
Pentachlorophenol	7.9×10^{-4}	No	0.03	Medium	Liver and Kidney Pathology	IRIS	Oral	100	1	2.6×10^{-2}	--	--
											0.23*	
Exposure Pathway:	**Dermal Contact with Soil**											
Arsenic	2.6×10^{-5}	Yes	0.001	--	Cancer, Keratosis	HEAST	Oral	1	--	2.6×10^{-2}	--	--
Chromium (Total, III)	4.1×10^{-5}	Yes	10	--	Hepatotoxocity	HEAST	Diet	100	1	4.1×10^{-6}	--	--
Chromium (VI)	5.2×10^{-7}	Yes	0.02	--	No effect	HEAST	Water	100	1	2.6×10^{-5}	--	--

02[UZ]ZD3081:D3123/4108/2

Key at end of table.

Table 8-32 (Cont.)

Chemical	CDI (mg/kg·day)	CDI Adj. for Absorp.	RfD (mg/kg·day)	Confidence Level	Critical Effect	RfD Source	RfD Basis	RfD Uncertainty Adj.	Modif. Factor	Hazard Quotient	Pathway Hazard Index	Total Exposure Hazard Index
Copper	$3.0x10^{-5}$	Yes	0.037	--	Local Irritation	HEAST	Water	--	--	$8.0x10^{-4}$	--	--
Pentachlorophenol	$1.0x10^{-3}$	Yes	0.03	Medium	Liver and Kidney Pathology	IRIS	Oral	100	1	$3.4x10^{-2}$	--	--
											$6.1x10^{-2}$*	
Exposure Pathway: Inhalation of Soil Particulate												
Arsenic	$1.9x10^{-6}$	No	0.001	--	Cancer, Keratosis	HEAST	Oral	1	--	$1.9x10^{-3}$	--	--
Chromium (Total, III)	$3.0x10^{-6}$	No	10	--	Hepatotoxicity	HEAST	Diet	100	1	$3.0x10^{-7}$	--	--
Chromium (VI)	$3.8x10^{-8}$	No	0.02	--	No effect	HEAST	Water	100	1	$1.9x10^{-6}$	--	--
Copper	$2.2x10^{-6}$	No	0.037	--	Local Irritation	HEAST	Water	--	--	$5.8x10^{-5}$	--	--
Pentachlorophenol	$7.6x10^{-6}$	No	0.03	Medium	Liver and Kidney Pathology	IRIS	Oral	100	1	$2.5x10^{-4}$	--	--
											$2.2x10^{-3}$*	
Child Residential soil exposures -- Total Subchronic Hazard Index												$3.0x10^{-1}$*

02[UZ]ZD3081:D3123/4108/2

Key:

-- = Not specified.
* = Does not total exactly due to independent rounding.
IRIS = Integrated Risk Information System.
HEAST = Health Effects Assessment Summary Tables.

Source: Ecology and Environment, Inc. 1991.

Table 8-33

CANCER RISK ESTIMATES
FUTURE USE
RESIDENTIAL GROUNDWATER USE, LOWER AQUIFER
REASONABLE MAXIMUM EXPOSURE - ADULT

Chemical	CDI (mg/kg-day)	CDI Adj. for Absorp.	SF (mg/kg-day)$^{-1}$	Weight of Evidence	Type of Cancer	SF Source	SF Basis (Vehicle)	Chemical-Specific Risk	Total Pathway Risk	Total Exposure Risk
Exposure Pathway: Ingestion of Drinking Water, Adult										
Pentachlorophenol	2.0×10^{-3}	No	0.12	B2	--	HEAST	Diet	2.4×10^{-4}	--	--
Exposure Pathway: Dermal Contact While Showering, Adult										
Pentachlorophenol	7.6×10^{-6}	Yes	0.12	B2	--	HEAST	Diet	9.2×10^{-7}	--	--
Exposure Pathway: Inhalation of Airborne Chemicals While Showering, Adult										
Pentachlorophenol	5.8×10^{-5}	No	0.12	B2	--	HEAST	Diet	7.0×10^{-6}	--	--
										2.4×10^{-4}

02[UZ]ZD3081:D3123/5044/6

Key:

-- = Not specified.
IRIS = Integrated Risk Information System.
HEAST = Health Effects Assessment Summary Tables.

*Does not total exactly due to independent rounding of chemical-specific and total pathway risks.

Source: Ecology and Environment, Inc. 1991.

Table 8-34

CANCER RISK ESTIMATES
FUTURE USE
RESIDENTIAL GROUNDWATER USE, UPPER AQUIFER
REASONABLE MAXIMUM EXPOSURE - ADULT

Chemical	CDI (mg/kg·day)	CDI Adj. for Absorp.	SF $(mg/kg \cdot day)^{-1}$	Weight of Evidence	Type of Cancer	SF Source	SF Basis (Vehicle)	Chemical-Specific Risk	Total Pathway Risk	Total Exposure Risk
Exposure Pathway: Ingestion of Drinking Water, Adult										
Pentachlorophenol	1.5×10^{-1}	No	0.12	B2	--	HEAST	Diet	1.8×10^{-2}	--	--
Exposure Pathway: Dermal Contact While Showering, Adult										
Pentachlorophenol	5.8×10^{-4}	Yes	0.12	B2	--	HEAST	Diet	6.9×10^{-5}	--	--
Exposure Pathway: Inhalation of Airborne Chemicals While Showering, Adult										
Pentachlorophenol	4.4×10^{-3}	No	0.12	B2	--	HEAST	Diet	5.3×10^{-4}	--	--
										1.8×10^{-2}

02[UZ]ZD3081:D3123/5045/6

Key:

-- = Not specified.
IRIS = Integrated Risk Information System.
HEAST = Health Effects Assessment Summary Tables.

*Does not total exactly due to independent rounding of chemical-specific and total pathway risks.

Source: Ecology and Environment, Inc. 1991.

Table 8-35

CHRONIC HAZARD INDEX ESTIMATES
FUTURE USE
RESIDENTIAL GROUNDWATER USE, LOWER AQUIFER
REASONABLE MAXIMUM EXPOSURE - ADULT

Chemical	CDI (mg/kg·day)	CDI Adj. for Absorp.	RfD (mg/kg·day)	Confidence Level	Critical Effect	RfD Source	RfD Basis	RfD Uncertainty Adj.	Modif. Factor	Hazard Quotient	Pathway Hazard Index	Total Exposure Hazard Index
Exposure Pathway: Ingestion of Drinking Water, Adult												
Pentachlorophenol	4.6×10^{-3}	No	0.03	Medium	Liver and kidney pathology	IRIS	Oral	100	1	1.5×10^{-1}	--	--
Exposure Pathway: Dermal Contact While Showering, Adult												
Pentachlorophenol	1.0×10^{-5}	Yes	0.03	Medium	Liver and kidney pathology	IRIS	Oral	100	1	3.4×10^{-4}	--	--
Exposure Pathway: Inhalation of Airborne Chemicals While Showering, Adult												
Pentachlorophenol	7.8×10^{-5}	No	0.03	Medium	Liver and kidney pathology	IRIS	Oral	100	1	2.6×10^{-3}	--	--
												1.6×10^{-1}

02[UZ]ZD3081:D3123/5046/0

Key:

-- = Not specified.
IRIS = Integrated Risk Information System.
HEAST = Health Effects Assessment Summary Tables.

*Does not total exactly due to independent rounding of chemical-specific and total pathway risks.

Source: Ecology and Environment, Inc. 1991.

Table 8-36

CHRONIC HAZARD INDEX ESTIMATES
FUTURE USE
RESIDENTIAL GROUNDWATER USE, UPPER AQUIFER
REASONABLE MAXIMUM EXPOSURE - ADULT

Chemical	CDI (mg/kg-day)	CDI Adj. for Absorp.	RfD (mg/kg-day)	Confidence Level	Critical Effect	RfD Source	RfD Basis	RfD Uncertainty Adj.	Modif. Factor	Hazard Quotient	Pathway Hazard Index	Total Exposure Hazard Index
Exposure Pathway: Ingestion of Drinking Water, Adult												
Pentachlorophenol	3.5×10^{-1}	No	0.03	Medium	Liver and kidney pathology	IRIS	Oral	100	1	1.2×10^{1}	--	--
Exposure Pathway: Dermal Contact While Showering, Adult												
Pentachlorophenol	7.8×10^{-4}	Yes	0.03	Medium	Liver and kidney pathology	IRIS	Oral	100	1	2.6×10^{-2}	--	--
Exposure Pathway: Inhalation of Airborne Chemicals While Showering, Adult												
Pentachlorophenol	5.9×10^{-3}	No	0.03	Medium	Liver and kidney pathology	IRIS	Oral	100	1	2.0×10^{-1}	--	--
												1.2×10^{1}

02[UZ]ZD3081:D3123/5047/0

Key:

-- = Not specified.
IRIS = Integrated Risk Information System.
HEAST = Health Effects Assessment Summary Tables.

*Does not total exactly due to independent rounding of chemical-specific and total pathway risks.

Source: Ecology and Environment, Inc. 1991.

For the exposure assessment, factors that would likely cause overestimation of the true exposures were:

- The use of the upper 95% confidence limit or highest observed values to estimate the soil and groundwater concentrations for RME estimates;
- The use of the steady state assumption for source concentration estimates.

The cumulative effect of all of the exposure uncertainties probably is to overestimate rather than underestimate the true potential exposures.

The basic uncertainties underlying the assessment of the toxicity of a chemical include:

- Uncertainties arising from the design, execution, or relevance of the scientific studies that form the basis of the assessment; and
- Uncertainties involved in extrapolating from the underlying scientific studies to the exposure situation being evaluated, including variable responses to chemical exposures within human and animal populations, between species and between routes of exposure.

These basic uncertainties could result in a toxicity estimate, based directly on the underlying studies, that either under- or overestimates the true toxicity of a chemical in the circumstances of interest.

The toxicity assessment process compensates for these basic uncertainties through the use of safety factors (uncertainty factors) and modifying factors, when assessing noncarcinogens, and the use of the upper 95% confidence limit from the linearized multistage model for the SF when assessing carcinogens. The use of the safety factors and the upper 95% confidence limit in deriving the RfDs and SFs ensures that the toxicity values used in the risk estimation process are very unlikely to underestimate, and hence almost always overestimate, the true toxicity of a chemical.

There are several additional factors that need to be considered when discussing uncertainties associated with the overall risk characterization. These are the cumulative effect of using conservative

assumptions throughout the process, and the likelihood of the exposures postulated and estimated in the exposure assessment actually occurring.

The cumulative effect of using conservative assumptions throughout the risk estimation process is that the resulting estimate will substantially overestimate the true risks. The Risk Assessment Guidance for Superfund manual (EPA 1989a) recommends that individual parameter values be selected so that the overall estimate of exposure, for example, represents a "reasonable maximum exposure." In many cases, the statistical distribution of a parameter is unknown and the risk assessor is left to select a value, using best professional judgment, that is sufficiently conservative to avoid underestimating the true risk, yet not so conservative that the resulting risk estimate turns out to be unreasonably high. When in doubt, the risk assessor will usually elect to err in favor of protecting human health and select a value that results in overestimating the true risk.

Conservative estimates are typically used at every stage of the risk assessment process, including:

- Selection or derivation of source media concentrations;
- Selection of the parameters used in estimating contaminant migration and receptor exposure;
- Selection or derivation of the reasonable maximum exposure point concentrations over the exposure duration postulated (steady state assumption is often used); and
- Derivation of quantitative indices of toxicity (safety factors are used in deriving RfDs, and the upper 95% confidence limit on the multistage model estimate is used as the carcinogenic potency SF).

In the risk estimation process, these estimates and the parameters contributing to the estimates are usually combined by multiplying them together. If two values, each an upper 95th percentile estimate, are multiplied together, the resulting value would be an upper 99.75th percentile estimate for the product. If three 95th percentile values are multiplied together, the result is an upper 99.98th percentile estimate, and four 95th percentile estimates yield a 99.999th percentile product,

which means the estimate has less than 1 chance in 100,000 of underestimating the actual value. A risk estimate derived in this way would obviously be extremely conservative and would substantially overestimate the true risks. There are many instances in the risk assessment process in which four or more parameters are multiplied together to obtain a risk estimate.

In summary, the nature of the risk estimation process itself virtually ensures that the true risks will be overestimated, sometimes by large margins. Many factors are multiplied together, inherent uncertainties exist about parameter values, and conscious decisions are made by risk assessors and the regulatory agencies to err on the side of protecting human health.

The last uncertainty factor to consider is the likelihood of the postulated exposures actually occurring. The exposure pathways identified as complete under current land use conditions are all plausible and exposure is either presently occurring by these pathways or such exposure could reasonably be expected. The postulated frequencies of occurrence may overestimate routine occurrence but could certainly reflect the reasonable maximum occurrence.

The first thing that must be addressed when considering the likelihood of exposure actually occurring by a potential future pathway is whether the postulated future land use is likely to occur. In this case, it was assumed that the Saunders property might be converted to residential use. The Saunders property is presently bounded by residential areas on three sides, indicating future residential use of the property is quite possible. Whether it is likely is another matter. The property has been an industrial site for about 25 years and has been owned and occupied by the current owners since 1946. While residential use of the Saunders property is certainly possible, it is probably not the most likely future use.

All soil-related exposure pathways identified as potentially complete under the future residential use scenario are quite plausible and could be expected to mediate potential future exposures. However, exposure to contaminants through use of site groundwater as a source of drinking water is unlikely since the city of Suffolk has a municipal water system that would be available to any new homes constructed on the site.

8.5.3 Summary Discussion of the Risk Characterization

This section will review and summarize the key elements of the risk assessment process for the Saunders site.

Characterization of Contamination Present at the Site

The remedial investigation was designed to characterize the nature, extent, and limits of contamination originating at the Saunders property and has successfully accomplished that goal. The possible source areas were identified based on a review of past and current industrial activities at the site. All of the possible source areas were then investigated using various field techniques and by collection and laboratory analysis of samples. In this way, the nature of the contamination was characterized and its extent defined.

Given the historical information available about the Saunders property it seems unlikely that any significant source areas were overlooked. Since samples were collected from the central parts of all source areas and, in most cases, were analyzed for the full TCL plus any non-TCL organics that were found, it is also unlikely that any significant contaminants would have been missed.

Major Factors Driving the Estimated Site Risks

The potential current risk to workers and future risks to residents, should the Saunders property be converted to residential use, would be due mainly to the proximity of the soil contaminants to the ground surface and the contaminants' toxicity. If groundwater were to be used by future residents as a drinking water source, they could face additional, potentially significant risks from exposure to PCP.

Characteristics of the Potentially Exposed Populations

Normal site activity is limited to moving dried treated lumber by forklift and loading it onto trucks. Presently, these positions are occupied by males. Saunders workers load and unload lumber from the ground using fork lifts and drive across the ground but have no direct work activities with soils on the Saunders property.

Potential future residents of the Saunders property would be expected to exhibit demographic characteristics typical of the City of Suffolk, Virginia.

Magnitude and Sources of Risks Posed by Site Contaminants

EPA has recently adopted the policy that acceptable exposures to known or suspected carcinogens are generally those that represent an excess upper bound lifetime cancer risk to an individual of between 10^{-4} and 10^{-6} (or one in 10,000 to one in 1,000,000). In addition, EPA will use the 10^{-6} risk level as the point of departure for determining remediation goals for NPL sites. For systemic toxicants (noncarcinogens) EPA defines acceptable exposure levels as those to which the human population, including sensitive subgroups, may be exposed without adverse effects during a lifetime or part of a lifetime, incorporating an adequate margin of safety (EPA 1990a). This acceptable exposure level corresponds to a hazard index of 1. If the hazard index is less than 1, no adverse effects would be expected. If the hazard index is greater than 1, adverse effects could be possible.

The magnitude of the potential carcinogenic risks posed by site contaminants is summarized in Table 8-37 and for current and future land use conditions. Tables 8-38 and 8-39 summarize the corresponding noncarcinogenic risks. Detailed information used to construct these tables is presented in Appendix K. Also included in these tables are the exposure routes and chemicals primarily responsible for the potential risks.

Under current land use conditions, total estimated cancer risks for workers by all three exposure routes--soil ingestion, soil dermal adsorption, and soil particulate inhalation--were 3.6×10^{-4}, exceeding EPA's acceptable range. Under potential future on-site residential use conditions, total cancer risks for all three soil exposure routes of 9.9×10^{-4} also exceeded the acceptable range. Table 8-37 displays the breakdown of percentage of total estimated cancer risks by exposure route. It is particularly noteworthy that soil ingestion dominates risks with over 70% of total risks attributed to this exposure route for both use conditions of the Saunders property.

Table 8-37

SAUNDERS HUMAN HEALTH RISK ASSESSMENT
SUMMARY OF ESTIMATED EXCESS LIFETIME CANCER
RISKS UNDER CURRENT AND FUTURE LAND USE CONDITIONS

Exposure Scenario	Receptor	Exposure Route	Cancer Risk	% of Total Cancer Risk	Chemicals Primarily Responsible for Cancer Risks in Order of Importance
Current On-Site Workers					
Outdoor Soil Exposures	Adult	Soil Ingestion	2.6×10^{-4}	74%	TCDD, Arsenic
	Adult	Soil Dermal Absorption	3.1×10^{-5}	8%	TCDD, Arsenic
	Adult	Soil Particulate Inhalation	6.3×10^{-5}	18%	Arsenic, TCDD
		Total	3.6×10^{-4}	100%	
Future On-Site Residential					
Outdoor Soil Exposures	Composite Lifetime	Soil Ingestion	7.7×10^{-4}	78%	TCDD, Arsenic, Pentachlorophenol
		Soil Dermal Absorption	1.8×10^{-4}	18%	TCDD, Pentachlorophenol, Arsenic
		Soil Particulate Inhalation	3.6×10^{-5}	4%	Arsenic, TCDD, Chromium (VI)
		Total	9.9×10^{-4}	100%	

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Table 8-37 (Cont.)

Exposure Scenario	Receptor	Exposure Route	Cancer Risk	% of Total Cancer Risk	Chemicals Primarily Responsible for Cancer Risks in Order of Importance
	Child	Soil Ingestion	3.5×10^{-4}	84%	TCDD, Arsenic, Pentachlorophenol
		Soil Dermal Absorption	5.4×10^{-5}	13%	TCDD, Pentachlorophenol, Arsenic
		Soil Particulate Inhalation	1.0×10^{-5}	3%	Arsenic, TCDD, Chromium (VI)
		Total	4.0×10^{-4}	100%	
Future On-Site Residential					
Groundwater Use Lower Aquifer	Adult	Water Ingestion	2.4×10^{-4}	97	Pentachlorophenol
		Water Dermal Absorption	9.2×10^{-7}	--	Pentachlorophenol
		Airborne Chemical Inhalation	7.0×10^{-6}	3	Pentachlorophenol
		Total	2.4×10^{-4}	100%	
Groundwater Use Upper Aquifer	Adult	Water Ingestion	1.8×10^{-2}	97	Pentachlorophenol
		Water Dermal Absorption	6.9×10^{-5}	--	Pentachlorophenol
		Airborne Chemical Inhalation	5.3×10^{-4}	3	Pentachlorophenol
		Total	1.8×10^{-2}	100%	

02[UZ]ZD3081:D3123/5048/14

Source: Ecology and Environment, Inc. 1991.

Table 8-38

SAUNDERS HUMAN HEALTH RISK ASSESSMENT
SUMMARY OF ESTIMATED NONCARCINOGENIC HAZARD INDICES
UNDER CURRENT LAND USE CONDITIONS

Exposure Scenario	Receptor	Exposure Route	Hazard Index	Chemicals Primarily Responsible for Risks in Order of Importance
Current On-Site Workers				
	Adult	Soil Ingestion	1.0×10^{-1}	Arsenic, Copper
		Soil Dermal Absorption	1.0×10^{-2}	Arsenic, Copper
		Soil Particulate Inhalation	2.1×10^{-3}	Arsenic, Copper
		Total	1.2×10^{-1}	

02[UZ]ZD3081:D3123/5049/I5

Source: Ecology and Environment, Inc. 1991.

Table 8-39

SAUNDERS HUMAN HEALTH RISK ASSESSMENT
SUMMARY OF ESTIMATED NONCARCINOGENIC HAZARD INDICES
UNDER FUTURE LAND USE CONDITIONS

Exposure Scenario	Receptor	Exposure Routes	Hazard Index	Chemicals Primarily Responsible for Risks in Order of Importance
Future On-Site Residential				
Outdoor Soil Exposures	Adult Male	Soil Ingestion	2.7×10^{-2}	Pentachlorophenol, Arsenic
		Soil Dermal Absorption	1.0×10^{-2}	Pentachlorophenol, Arsenic
		Soil Particulate Inhalation	5.1×10^{-4}	Arsenic, Pentachlorophenol, Copper
		Total	4.0×10^{-2}	
	Child	Soil Ingestion	2.3×10^{-1}	Arsenic, Pentachlorophenol
		Soil Dermal Absorption	6.1×10^{-2}	Pentachlorophenol, Arsenic
		Soil Particulate Inhalation	2.2×10^{-3}	Arsenic, Pentachlorophenol
		Total	3.0×10^{-1}	
Groundwater Use Lower Aquifer	Adult	Water Ingestion	1.5×10^{-1}	Pentachlorophenol
		Water Dermal Absorption	3.4×10^{-4}	Pentachlorophenol
		Airborne Chemical Inhalation	2.6×10^{-3}	Pentachlorophenol
		Total	1.6×10^{-1}	
Groundwater Use Upper Aquifer	Adult	Water Ingestion	1.2×10^{-1}	Pentachlorophenol
		Water Dermal Absorption	2.6×10^{-2}	Pentachlorophenol
		Airborne Chemical Inhalation	2.0×10^{-1}	Pentachlorophenol
		Total	1.2×10^{-1}	

02[UZ]ZD3081:D3123/5050/14

Source: Ecology and Environment, Inc. 1991.

For potential future residential use of groundwater, cancer risk estimates are presented separately for the lower aquifer and the upper aquifer. In both cases the total estimated cancer risks from all three routes exceeded the acceptable 10^{-4} to 10^{-6} range. Exposures by ingestion of drinking water accounted for 97% of the total estimated cancer risk due to groundwater use.

None of the exposure pathways that are complete under current land use conditions were estimated to result in potential exposures to non-carcinogenic contaminants that might produce adverse health effects. The total estimated hazard index (Table 8-38) was 1.2×10^{-1}, which is approximately one eighth the benchmark level of 1, above which some adverse effects might be expected.

Table 8-39 indicates that potential exposure to PCP through groundwater usage could also result in adverse noncarcinogenic health effects should the Saunders property be converted to residential use without prior remedial measures. Table 8-39 indicates that potential future residential use of groundwater containing the highest PCP concentrations observed in the Yorktown aquifer would not pose a significant risk of adverse noncarcinogenic effects (Hazard Index <1). However, if the PCP concentration in the Yorktown aquifer rose as high as those observed in the Columbian water-bearing zone, and the water was used for domestic purposes, adverse noncarcinogenic effects could occur (Hazard Index = 12). With the availability of public water via the municipal supply system, the use of groundwater as a drinking water source is very unlikely, even if the site were converted to residential use.

Exposure to soil contaminants only would not be expected to have noncarcinogenic adverse health effects; the total hazard indices for both adult and child receptors was less than 1.

Nature of Potential Adverse Health Effects

Based on the above results the only chemicals contributing to potentially significant adverse health effects under current land use conditions are arsenic, PCP, and dioxin/furans. Arsenic is considered a human carcinogen based on epidemiological studies in workers occupationally exposed to this chemical; whereas PCP and dioxin have produced cancer in rodents.

The same chemicals could contribute significantly to potential adverse health effects if the Saunders property were to be converted to residential use without prior remedial measures being taken. The adverse health effects of these chemicals are described in Appendix K. They are also included in Tables 8-25 and 8-26, along with information on critical effects and the strength of the evidence supporting the toxicological assessments.

Level of Confidence/Uncertainty in the Risk Estimates

As discussed fully in earlier Sections (8.3.9, 8.5.2) of this report, the level of confidence in the exposure estimates is moderate to good. The level of confidence in the toxicity estimates varies from chemical to chemical as shown in Tables 8-25 and 8-26.

Overall, the level of confidence in the risk estimates are also moderate to good. However, as noted earlier, the nature of the risk assessment process strongly favors overestimation of the true risks. Accordingly, the risk estimates presented here are quite likely to overestimate the true risks but unlikely to underestimate them.

9. ECOLOGICAL ASSESSMENT

9.1 INTRODUCTION AND METHODS

The purpose of the ecological assessment is to determine if contaminants related to the Saunders wood treating facility are present in nearby surface waters and sediments in available concentrations sufficient to cause adverse ecological impacts. As part of the ecological assessment, risks to the ecological environment are addressed. The approach taken in the ecological risk assessment is based on EPA (1989e, 1989f). Section 1 of this report should be referred to for information on the background history and description of the Saunders facility.

A wide variety of tools for detecting the effects of chemical stress on biological communities were evaluated for applicability to this assessment. Table 9-1 summarizes commonly used biological parameters and evaluates their usefulness for this study. Upon evaluation of the expense, level of effort, and suitability of each parameter, relative to available resources and knowledge of site conditions prior to the initiation of this study, appropriate parameters and ecological endpoints were chosen for measurement. A summary of these is provided in Table 9-2. The methods and results of the evaluation of these endpoints have already been described in Sections 2.9, 3.11, and 5.6. The results of the ecological assessment can be used to support the development of appropriate cleanup goals for the Saunders study area.

The specific objectives and organization of this assessment are as follows.

Table 9-1

BIOLOGICAL PARAMETERS FOR ECOLOGICAL ASSESSMENT OF CHEMICAL STRESS

Parameter	Effect Observed in Stressed vs. Unstressed System	Use Warranted?
1. Indicator Species	Absence of sensitive species	No
2. Physiological Condition	Gross pathology (tumors, lesions, wilted foliage, etc.)	Yes
3. Biomass and Abundance	Some elements of biota lower, some higher	Yes
4. Biotic Indices	Systematic difference	No
5. Species Richness	Usually lower	No
6. Species Diversity	No reliable, systematic effect	No
7. Single-species toxicity bioassay	Toxic effects on survival, reproduction, or growth	Yes
8. Biomarkers	Altered enzyme activity, DNA abnormalities, tissue residues of contaminants, histopathological and skeletal abnormalities, physiological dysfunction	No

02[UZ]ZD3081:D3123/4087/23

Source: EPA 1989b; Ford 1989.

Table 9-2

MEASUREMENT ENDPOINTS UTILIZED FOR ECOLOGICAL ASSESSMENT OF THE SAUNDERS SITE

Parameter	Endpoints	Measurement
Physiological condition	Fish tumors Fish reproduction Stressed vegetation	Visual or photographic evidence of presence or absence
Biomass and abundance	Fish community Macrobenthos Vegetation	Qualitative assessment of relative abundance and species composition
Single-species toxicity bioassay	*Daphnia magna*: Survival and reproduction	Number of survivors and number of progeny
	Chironomus tentans: Survival and growth	Number of survivors, weight and length

02[UZ]ZD3081:D3123/4088/19

Source: Ecology and Environment, Inc. 1991.

Exposure Analysis (Section 9.2)

- o Identify contaminants of concern;
- o Identify environmental receptors, i.e., populations and communities potentially exposed to the contaminants of concern; and
- o Derive exposure concentrations, or expected environmental concentrations (EECs) for contaminants of concern.

Toxicity Assessment (Section 9.3)

- o Review the available toxicological literature on contaminants of concern; and
- o Derive benchmark criteria (BC) and Environmental Concern Levels (ECLs) for contaminants of concern.

Ecological Risk Assessment (Section 9.4)

- o Assess the baseline risk to the potentially exposed biota under the no-action alternative; and
- o Evaluate evidence of adverse ecological effects related to the site, based on the relationship between results of chemical analyses, toxicity tests, benthic surveys, and exposure/toxicity assessment (EPA 1989e; 1989f).

9.2 EXPOSURE ANALYSIS

Identification of Contaminants of Concern

For the purposes of the ecological assessment, contaminants generated by CCA wood preservation facilities (Konasewich and Henning 1988) or PCP wood preservation facilities (Eisler 1989) were considered to be the contaminants of concern. Arsenic, chromium (total), chromium VI, copper, dioxin/furans, and PCP were identified as contaminants of concern, based on the literature and information on site contaminants provided in Section 6.

Identification of Environmental Receptors

Environmental receptors are populations and communities of organisms potentially exposed to contamination. For the purposes of the risk assessment, a subset of environmental receptors were chosen to serve as

biotic focal elements for analysis. Criteria for selection of particular species or groups of species as focal elements are as follows:

- Focal elements of the biota are species or communities of intrinsic importance for economic or recreational reasons, or for regulatory reasons (e.g., endangered species), or that could serve as vectors for human exposure;
- Focal elements are known to provide an early warning signal of potential effects, or are particularly and reliably sensitive to chemical stress;
- Focal elements are known to play a critical ecological role in the food chain, or are indicative of alterations in ecosystem processes such as energy flow or nutrient cycling; and
- Focal elements are representative of or are known to occur in habitats potentially affected by contamination.

The Virginia Department of Conservation and Recreation's Division of Natural Heritage database contained no records of natural heritage resources, such as rare species or exemplary natural communities, in the study area. Records of three aquatic species were found within a 15-mile surface water range of the Saunders property. These include the lined topminnow, the sawcheek darter, and the Tidewater amphipod (see Section 2.9.2).

Given the absence of recorded observations for these species within the study area, no effort was made to consider them explicitly in the risk assessment. However, the approach taken (see below) to protect aquatic life should extend a degree of safety for these species, should they occur in the area.

In addition, the Virginia Department of Game and Inland Fisheries reported no wilderness areas, natural areas, or scenic rivers in the immediate vicinity of Chuckatuck, Virginia. Three wildlife refuges (Nansemond, Dismal Swamp, and Back Bay) are in the general area, however, and Dismal Swamp Wildlife Refuge borders the Chuckatuck Quad. These wildlife refuges are not close enough to Saunders to be impacted by site contaminants, but they could conceivably provide source populations of critical species to colonize the vicinity of the project area. In addition, three special status terrestrial species are known

that may exist in the vicinity of the Suffolk and Chuckatuck Quads, according to the Department. These include the bald eagle, the rafinesque's big-eared bat, and the Dismal Swamp southeastern shrew (see Section 2.9.3).

Bald eagles may be observed in the area but none are known to nest in or near Chuckatuck Quad. Rafinesque's big-eared bat occurs near Dismal Swamp but has not been reported from Chuckatuck Quad. The Dismal Swamp southeastern shrew has been reported from Chuckatuck Quad (Kitchel 1991).

It is unlikely that the Dismal Swamp southeastern shrew is located within the confines of the Saunders property given the industrial activity associated with the wood treatment operations. The site would need to be surveyed by a specialist to determine the presence or absence of this shrew on the Saunders property.

The primary contaminant exposure routes to the environment are through the aquatic ecosystem. Therefore the ecological assessment detailed aquatic species exposure to contaminants derived from the Saunders property. Terrestrial exposure routes would be considered if terrestrial species, such as the Dismal Swamp southeastern shrew were documented to exist on the site.

Fish populations in Godwin's Millpond are of recreational importance to local residents, although no single species could be considered to be of commercial significance. Therefore, this risk assessment considers the community of lacustrine, aquatic life as focal elements of the biota. This approach is intended to protect fish populations from the direct effects of toxic concentrations of contaminants, as well as from the effects of contaminants on zooplankton, phytoplankton, and benthos, the food sources of the fish. Observations of terrestrial species encountered during the field survey did not suggest the potential of contaminant effects upon this element of the ecological community.

Derivation of Exposure Concentrations

Exposure assessment involves the determination of EECs of the contaminants of concern, in space and time, at the interface with environmental receptors. On the basis of E & E's field reconnaissance,

which indicated little evidence of pronounced ecosystem dysfunction (Section 3.11), the time and expense of developing a site-specific fate and transport computer model were not justified. Instead, EECs were developed, based on the ambient concentrations of contaminants as measured in field samples and presented in Section 5. EECs were then derived from realistic exposure scenarios for each of the selected focal elements of the biota (e.g., fish exposed to ambient concentrations in the water column).

The Saunders wood treating facility itself does not represent significant wildlife habitat because of its nature as an industrial area. In addition, contaminant levels in habitats from the surface water bodies that drain the site are likely to reflect exposure concentrations for aquatic organisms more accurately than contaminant levels at the facility. Therefore, available measurements of chemical contamination in water and sediment of these habitats were used to calculate EECs.

Exposure analysis was limited to the surface water column. It was not possible to conduct exposure analysis for sediment-bound metals because the bioavailability of contaminants was not known. In general, for benthic organisms the sediment pore water concentration of contaminants is a better measure of available toxins than total sediment concentrations. Poor water concentration of non-polar organics can be estimated using the equilibrium partitioning (EP) approach (EPA 1989i). Hence, this method would be appropriate to derive EECs for sediments with PCP and dioxin/furan contaminants. For metals, however, there are no generally accepted methods for deriving pore water concentrations using the EP or other approaches.

The RAGs Human Health Evaluation Manual recommends developing both an "average exposure" and a "reasonable maximum exposure." This is the procedure followed in Tables 9-3 and 9-4, using media-specific concentrations of contaminants as measured at each location.

For the lacustrine aquatic community, a realistic worst case exposure scenario would involve continuous lifetime exposure to average levels of contaminants of concern found in Godwin's Millpond. Short duration acute exposure to the areas of greatest contamination could also be considered as a realistic worst case. Additional exposure could

Table 9-3

ESTIMATED ENVIRONMENTAL EXPOSURE CONCENTRATIONS (EECs)
FOR SHORT DURATION (ACUTE) EXPOSURES

Location[1]	Chemical	Environmental Exposure Concentrations (µg/L)[2]
Godwin's Millpond (GW-1, GW-2, GW-3 GW-5, GW-6)	Arsenic[3]	<10
	Chromium	<10
	Chromium VI	<10
	Copper	NQ
	PCP	<50
Cedar Creek (CE-1, CE-2, CE-3)	Arsenic	15.0
	Chromium	<10
	Chromium VI	<10
	Copper	NQ
	PCP	<50
Chuckatuck Creek (CK-1, CK-2, CK-3)	Arsenic	23.0
	Chromium	<10
	Chromium VI	<10
	Copper	NQ
	PCP	<50
Intermittent Stream (IS-1, IS-2, IS-3)	Arsenic	6.5
	Chromium	<10
	Chromium VI	<10
	Copper	NQ
	PCP	<50
Reference Site (GW-4)	Arsenic	<10
	Chromium	2.4
	Chromium VI	<10
	Copper	NQ
	PCP	<50

02[UZ]ZD3081:D3123/4089/29

Key:

[1]See Figure 5-12 for locations.

[2]Short duration (acute) ECCs were estimated to be the highest measured concentration of any sample at a given location; if duplicate samples were taken, the average was used.

[3]Arsenic levels are dissolved fraction; other metals are non-dissolved fraction.

NQ = Not quantifiable (see Section 5.3 of text).

Source: Ecology and Environment, Inc. 1991.

Table 9-4

ESTIMATED ENVIRONMENTAL EXPOSURE CONCENTRATIONS (EECs)
FOR LONG DURATION (CHRONIC) EXPOSURES

Location[1]	Chemical	Environmental Exposure Concentrations (µg/L)[2]
Godwin's Millpond (GW-1, GW-2, GW-3 GW-5, GW-6)	Arsenic[3]	<10
	Chromium	<10
	Chromium VI	<10
	Copper	NQ
	PCP	<50
Cedar Creek (CE-1, CE-2, CE-3)	Arsenic	13.0
	Chromium	<10
	Chromium VI	<10
	Copper	NQ
	PCP	<50
Chuckatuck Creek (CK-1, CK-2, CK-3)	Arsenic	11.0
	Chromium	<10
	Chromium VI	<10
	Copper	NQ
	PCP	<50
Intermittent Stream (IS-1, IS-2, IS-3)	Arsenic	5.3
	Chromium	<10
	Chromium VI	<10
	Copper	NQ
	PCP	<50
Reference Site (GW-4)	Arsenic	<10
	Chromium	2.4
	Chromium VI	<10
	Copper	NQ
	PCP	<50

02[UZ]ZD3081:D3123/4091/29

Key:

[1]See Figure 3-6 for Locations.

[2]Long duration (chronic) EECs were estimated to be the average of measured concentration for all samples at a given location. If some but not all of the samples were ND, the value for that sample was taken to be the average of 0.0 and the detection limit for that compound; if duplicate samples were taken the average was used.

[3]Arsenic levels are dissolved fraction; other metals are non-dissolved fraction.

NQ = Not quantifiable (see Section 5.3 of text).

Source: Ecology and Environment, Inc. 1991.

arise from biomagnification of these contaminants through the food chain. For certain individuals, the chronic exposure concentrations could be higher or lower depending on the spatial distribution of populations. Therefore, the exposure scenario was designed to represent an approximation of average effects on aquatic populations.

EECs for contaminants of concern (except for dioxin/furans) are given in Table 9-3 and Table 9-4. With the exception of arsenic, nearly all contaminants of concern were not found at EECs (short and long exposures) above detection limits in surface water samples. Copper concentrations were not quantifiable due to the inability of distinguishing any difference between samples and blanks (see Section 5.3). Chromium was found at an EEC above detection limits at only one location, the Reference Site (GW-4).

Dioxin/furans sampling in the surface water was conducted on a smaller subset of samples, which did not include any of the locations off site. Hence, dioxin/furans contamination of surface water is not considered in the ecological assessment, and no EECs for dioxin/furans were derived.

Since there was no evidence of impacts from the field survey of the terrestrial environment, and because the primary pathway for migration of contaminants is through surface runoff and leaching of groundwater to the adjacent aquatic ecosystems, a detailed scenario for terrestrial receptors was not developed.

In addition, since none of the metals identified as contaminants of concern bioaccumulate to a significant degree in freshwater organisms, and since neither PCP nor dioxin/furans were identified in sediment or surface water samples from aquatic ecosystems, the potential for exposure of fish-eating birds and mammals through their food was considered negligible.

9.3 TOXICITY ASSESSMENT

Review of Toxicological Literature

Information concerning the toxicity of the contaminants of concern was reviewed using a wide variety of literature resources.

Brief toxicological profiles of arsenic, chromium (total), chromium VI, copper, and PCP are found in Appendix K, which includes information relevant to both human and wildlife receptors.

Derivation of Benchmark Criteria and Environmental Concern Levels

Toxicological BC were derived for focal elements of the biota based on the literature review. BCs are toxicological indices of effects, usually based on laboratory bioassays of single species exposed to single toxic compounds. A threshold concentration for significant effects on survival, growth, or reproduction such as the maximum acceptable toxicant concentration (MATC) is usually used as a benchmark for chronic toxic effects. The MATC is assumed to lie between the no-observed effect level (NOEL) and the lowest-observed-effect level (LOEL) for a given contaminant (Suter 1986). The standard BC for acute toxic effects is the lethal concentration for 50% of the population (LC_{50}).

Toxicological benchmarks are frequently modified to provide assessment endpoints, in order to account for uncertainties in extrapolating from laboratory data to field situations, by multiplying the BC by an uncertainty factor ranging from 1.0 to 0.001. Uncertainty factors used to modify BCs and their rationale are shown in Table 9-5. The product of a BC multiplied by an uncertainty factor is an assessment endpoint termed the Environmental Concern Level (ECL).

The derivation of BC and ECLs for environmental receptors was based on published EPA Ambient Water Quality Criteria (AWQC) (see Table 9-6 and Table 9-7). Acute AWQC are based on LC50s or EC50s, and chronic AWQC are based on MATCs. The EPA AWQC are intended to protect 95% of aquatic species, and should therefore indicate appropriate ECLs for the aquatic organisms of Godwin's Millpond, with uncertainty factors = 1.0.

9.4 RISK ASSESSMENT

Evaluation of Baseline Risk

The evaluation of baseline risk to potentially exposed biota was based on application of the quotient method (Suter 1986).

Table 9-5

UNCERTAINTY FACTORS AND THEIR APPLICATION

Uncertainty Factor	Application to ECLs
0.10 - 1.0	Used when MATC (or NOEL) data is unavailable and LOEL data is used instead. The range of the factor depends on the reliability and appropriateness of the data and the experimental design.
0.010	Used when data for the biota of concern are unavailable but valid long-term studies for other species are used.
0.0010 - 0.010	Used when data for the biota of concern are unavailable and studies of less than chronic exposure are used. The magnitude of the factor depends upon the data and experimental design.
Intermediate Factors	Other uncertainty factors may be used based on scientific judgment.

02[UZ]ZD3081:D3123/4090/24

Source: Dourson and Stara 1983; Barnes and Dourson 1988; EPA 1984.

Table 9-6

ENVIRONMENTAL CONCERN LEVELS FOR AQUATIC ORGANISMS,
ACUTE EXPOSURE

Chemical	Selected Acute Benchmark Criteria (µg/L)[1]	Uncertainty Factor[2]	Acute ECL (µg/L)[3]
Arsenic[4]	800	1.0	800
Chromium[5,6]	980	1.0	980
Chromium VI	16	1.0	16
Copper[6]	9.2	1.0	9.2
PCP[7]	5.1	1.0	5.1

02[UZ]ZD3081:D3123/4092/30

Key:

[1]Value based on Ambient Water Quality Criteria for protection of freshwater aquatic organisms.

[2]See text.

[3]ECL = BC x Uncertainty factor.

[4]Arsenic V.

[5]Chromium III.

[6]Hardness dependent criteria; value given is for hardness 50 mg/L as $CaCO_3$.

[7]pH-dependent criteria; value given is for pH 6.5.

Sources: EPA 1985a,b,c; 1986; 1988.

Table 9-7

ENVIRONMENTAL CONCERN LEVELS FOR AQUATIC ORGANISMS,
CHRONIC EXPOSURE

Chemical	Selected Chronic Benchmark Criteria ($\mu g/L$)[1]	Uncertainty Factor[2]	Chronic ECL[3] ($\mu g/L$)
Arsenic[4]	48	1.0	48
Chromium[5,6]	120	1.0	120
Chromium VI	11	1.0	11
Copper	6.5	1.0	6.5
PCP[7]	3.2	1.0	3.2

02[UZ]ZD3081:D3123/4093/29

Key:

[1]Value based on Ambient Water Quality Criteria for protection of freshwater aquatic organisms.

[2]See text.

[3]ECL = BC x Uncertainty factor.

[4]Arsenic V.

[5]Chromium III.

[6]Hardness dependent criteria; value given is for hardness 50 mg/L as $CaCO_3$.

[7]pH-dependent criteria; value given is for pH 6.5.

Sources: EPA 1985a,b,c; 1986; 1988.

The quotient method is a standard approach for screening sample locations for potentially toxic concentrations of chemicals. It involves calculating the ratio of each EEC to the corresponding ECL. The higher the ratio is (greater than 1.0), the higher the probability of significant risk to the receptor population.

The comparison of ECLs to EECs for each of the contaminants of concern is given in Table 9-8, for acute exposures, and Table 9-9 for chronic exposures. All of the ratios are less than one, for arsenic, chromium, and chromium VI, indicating exposures below concern levels for these contaminants at all locations. Copper concentrations were not quantifiable, as discussed earlier; therefore the ratio was not determined. As for PCP, the detection level of 50 µg/L was higher than the acute or the chronic ECL for this compound; therefore it is not possible to determine whether the high ratio (>1) for this contaminant indicates a significant risk to the biota. Any levels above the detection limit, had they been found, would have indicated serious contamination, since the detection limit was nearly 10 times greater than the acute ECL (see Table 9-8).

On the basis of the quotient method, there does not appear to be a significant risk to aquatic life in Godwin's Millpond or adjacent streams from the levels of arsenic, chromium, and chromium VI in the surface water. It is not possible to make a determination of the risk, if any, from levels of copper and PCP in surface water.

Although it is not possible to quantify the risk from levels of copper, high concentrations of this contaminant were found in runoff water samples RW-1 (181 µg/L) and RW-2 (207 µg/L) (see Section 5.3). These levels could present a risk to aquatic life, since they significantly exceed both chronic and acute ECLs.

The main points of further consideration for the risk assessment are as follows:

- The presence of oily film and odor in sediments;
- The presence of a tumor on one of the fish collected from Godwin's Millpond;
- The low incidence of invertebrates collected from Godwin's Millpond;

Table 9-8

EEC/ECL RATIOS FOR AQUATIC ORGANISMS,
ACUTE EXPOSURE

Location	Chemical	Acute EEC ($\mu g/L$)[1]	Acute ECL ($\mu g/L$)[2]	EEC/ECL[3] Ratio
Godwin's Millpond	Arsenic	<10	800	<0.01
	Chromium	<10	980	<0.01
	Chromium VI	<10	16	<0.63
	Copper	NQ	9.2	NQ
	PCP	<50	5.1	<9.8
Cedar Creek	Arsenic	15.0	800	0.02
	Chromium	<10	980	<0.01
	Chromium VI	<10	16	<0.63
	Copper	NQ	9.2	NQ
	PCP	<50	5.1	<9.8
Chuckatuck Creek	Arsenic	23.0	800	0.03
	Chromium	<10	980	<0.01
	Chromium VI	<10	16	<0.63
	Copper	NQ	9.2	NQ
	PCP	<50	5.1	<9.8
Intermittent Stream	Arsenic	6.5	800	0.01
	Chromium	<10	980	<0.01
	Chromium VI	<10	16	<0.01
	Copper	NQ	9.2	NQ
	PCP	<50	5.1	<9.8
Reference Site	Arsenic	<10	800	<0.01
	Chromium	2.4	980	<0.01
	Chromium VI	<10	16	<0.63
	Copper	NQ	9.2	NQ
	PCP	<50	5.1	<9.8

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Key:

NQ = Not quantifiable (see Section 5.3 of text).

[1]Acute EECs are from Table 9.3.

[2]Acute ECLs are from Table 9.6.

[3]Ratio is unitless, rounded to two significant digits.

Source: Ecology and Environment, Inc., 1991.

Table 9-9

EEC/ECL RATIOS FOR AQUATIC ORGANISMS,
CHRONIC EXPOSURE

Location	Chemical	Chronic EEC (μg/L)[1]	Chronic ECL (μg/L)[2]	EEC/ECL[3] Ratio
Godwin's Millpond	Arsenic	<10	48	<0.21
	Chromium	<10	120	<0.08
	Chromium VI	<10	11	<0.91
	Copper	NQ	6.5	NQ
	PCP	<50	3.2	<15.6
Cedar Creek	Arsenic	13.0	48	0.27
	Chromium	<10	120	<0.08
	Chromium VI	<10	11	<0.91
	Copper	NQ	6.5	NQ
	PCP	<50	3.2	<15.6
Chuckatuck Creek	Arsenic	11.0	48	0.23
	Chromium	<10	120	<0.08
	Chromium VI	<10	11	<0.91
	Copper	NQ	6.5	NQ
	PCP	<50	3.2	<15.6
Intermittent Stream	Arsenic	5.3	48	0.11
	Chromium	<10	120	<0.08
	Chromium VI	<10	11	<0.91
	Copper	NQ	6.5	NQ
	PCP	<50	3.2	<15.6
Reference Site	Arsenic	<10	48	<0.21
	Chromium	2.4	120	0.02
	Chromium VI	<10	11	<0.91
	Copper	NQ	6.5	NQ
	PCP	<50	3.2	<15.6

02[UZ]ZD3081:D3123/4095/20

Key:

NQ = Not quantifiable (see Section 5.3 of text).

[1]Chronic EECs are from Table 9-3.

[2]Chronic ECLs are from Table 9-5.

[3]Ratio is unitless, rounded to two significant digits..

Source: Ecology and Environment, Inc., 1991.

- The spatial pattern of chemical contamination of sediments; and
- Chronic toxicity of sediments as indicated in bioassays.

An oily film and odor were noted along the intermittent stream upstream and downstream of the Saunders property, and at all sample locations on Godwin's Millpond. The widespread occurrence of this apparent contamination indicates the presence of source(s) other than Saunders. A historical contribution of Saunders to the problem cannot be conclusively ruled out, nor can the Saunders wood treating facility be definitively implicated as contributing to this apparent contamination. Boating activities and machinery on adjacent roads and agricultural fields are potential sources of petroleum-based products that would cause an oily film. On the other hand, the total petroleum hydrocarbons found in sediments of the intermittent stream could be related to the historical practice of spraying waste petroleum sludge on the site for weed and dust control (see Section 1). However, petroleum compounds and byproducts were not detected in surface water samples from Godwin's Millpond. Therefore, the baseline risk from this source of contamination can be presumed to be low, although oil pollution could be a contributing factor in sediment toxicity (see below).

The presence of a tumor on a bass collected from Godwin's Millpond is a possible indication of chemical contamination. A definitive link of the gross pathology of the fish tumor to contaminants of concern related to Saunders cannot be made, however, since a reasonably healthy fish population was noted in the field survey, and concentrations of contaminants of concern were not found at toxic levels in the surface water of aquatic habitats.

The paucity of invertebrates collected in the intermittent stream and Godwin's Millpond could be partly related to lack of suitable habitat, but the contribution of chemical contamination is implicated by:

- The results of sediment toxicity bioassay, showing chronic reproductive toxicity for Daphnia magna at all sample locations (Section 5.6); and

- The fact that very few aquatic invertebrates were collected. Aquatic invertebrates are sensitive to environmental contaminants, and their low abundance and diversity is an indicator of polluted conditions (Ford 1989).

But, as with the fish tumor, this indication of chemical stress cannot be definitively linked to Saunders, since concentrations of contaminants of concern were not found at toxic levels in surface water. Levels of these contaminants could be at toxic levels in sediment samples, however.

Guidelines for sediment pollution in the Great Lakes have been published (Great Lakes Water Quality Board 1982), and these were used to examine qualitatively if sediment-bound contaminants of concern were at levels indicative of polluted waters. The data are shown in Table 9-10. With the exception of one sample exceeding the guidelines for chromium, arsenic was the only contaminant showing concentrations above the recommended limits. It should be noted, however, that the arsenic limits appear to be set at a level below that typically found in lake sediments. A range of 5 to 26.9 mg/kg arsenic for lacustrine sediments is provided by Eisler (1988), for example. Only samples GW-6 (38.0 mg/kg) and CK-3 (72.0 mg/kg) exceeded this range. It would not be expected that the release of arsenic from CCA wood preservation at Saunders would contaminate sediments at GW-6, which is upstream from the Saunders wood treating facility. Local agricultural utilization of arsenical pesticides might be a more likely source of arsenic contamination at this location. Although a historical contribution of Saunders to the high arsenic concentration at the bottom of Chuckatuck Creek (CK-3) cannot be conclusively ruled out, other sources (such as agriculture and influx from tidal influence) could be equally or more important contributors to the apparent contamination.

Finally, the universal distribution of chronic toxic effects in sediment samples from all locations, including the reference site (GW-4), indicates widespread sediment contamination (see Section 5.6). Toxicity of sediment was observed in both elutriate and solid phase tests with Daphnia magna, but the sediment was not toxic to Chironomus tentans (Section 5.6). D. magna and C. tentans could have differing sensitivities and exposures to toxic chemicals and would not be expected

Table 9-10

COMPARISON OF SEDIMENT CONTAMINATION
TO REGULATORY GUIDELINES

Chemical	Concentration Limits[1] (mg/kg)	Samples Exceeding Lower Limit[2]	Samples Exceeding Upper Limit
Arsenic	3 - 8	IS-1, GW-2	GW-6, CK-2, CK-3
Chromium	25 - 75	CK-3	None
Chromium VI	NA	ND	ND
Copper	26 - 50	None	None
PCP	NA	ND	ND

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Key:

NA = Limits not available
ND = Not detected

[1]Limits define the range for moderately polluted sediments; concentration below the lower limit are considered non-polluted, concentrations above the upper limit are considered heavily polluted (Great Lakes Water Quality Board 1982).

[2]See Figure 5-12 for sample locations.

Source: Ecology and Environment, Inc. 1991.

to have parallel responses in toxicity tests. Therefore, the results of the laboratory bioassays demonstrate the potential for adverse effects of sediments on some invertebrates, though it is not known if the particular species which are present in Godwin's Millpond (see Table 4-5) are adversely affected by sediment contamination. Due to the lack of significant differences between reference and other samples, the toxicity of sediments cannot be attracted to the Saunders wood treatment facility. In addition, the spatial distribution of contaminants of concern in sediment samples is not correlated with this pattern of toxic effects, since only the two locations mentioned above are contaminated with arsenic. Thus, as with the oily film and odor discussed earlier, the extent of contamination indicates a source(s) of contamination other than Saunders. Agricultural and waste disposal activities in the area are possible contributors to the contamination, but it is not possible to identify any source in particular from the available data.

In summary, the ecological assessment has found evidence suggestive of the potential for adverse ecological impacts in sediments of Godwin's Millpond and adjacent intermittent stream aquatic habitats, based on laboratory toxicity tests and the low abundance of aquatic macroinvertebrates. The spatial extent of this contamination indicates a source or sources other than Saunders, however, and the spatial distribution of contaminants of concern in surface waters or sediments does not provide any evidence that contaminants related to Saunders are the causal agent of adverse ecological impacts. The status of aquatic life in the area suggests that the land use associated with the watershed and perhaps the metal contamination is having a moderate effect on ecosystem health, but fish populations of recreational importance appear to be adequate despite the contamination.

10. SUMMARY AND CONCLUSIONS

10.1 SUMMARY OF FINDINGS

The RI for the Saunders Supply Company, located in Chuckatuck, Virginia, was performed by E & E, under contract to EPA Region III. RI field activities were conducted from May 1989 through November 1990. The primary purpose of the RI was to assess the extent of contamination, characterize the factors affecting the movement of the contaminants, and evaluate the threat that the contaminants may pose to public health and the environment. In addition, the information obtained will allow for the development of remedial action alternatives as part of the FS.

The field investigations focused primarily within the Saunders property boundaries, portions of the adjacent Kelly property, as well as on downgradient areas between the Saunders property and Godwin's Millpond. Godwin's Millpond is a public drinking water source for the City of Suffolk, Virginia.

Prior to sampling of environmental media, preliminary field activities were performed. These activities consisted of a topographic survey, an X-ray fluorescence survey, and a geophysical survey. The topography of the Saunders property is characterized by a slight topographic rise, which begins at the southwestern portion of the Saunders property along Crumpler Lane and ends in the vicinity of the former conical burn pit. Surface drainage east of this rise discharges primarily to the north with the portion farthest east of the site draining to the east into a series of catch basins associated with the storm sewer system of Route 10/32. Drainage collected by the sewer system at this location is discharged to a surface swale, which ultimately discharges to Cedar Creek. On the west side of the topographic rise, surface drainage from the Saunders property flows west and

is either intercepted by the existing wastewater pond or flows directly into an intermittent stream, which in turn flows into Godwin's Millpond.

A grid was established across the site during the topographic survey to assist the X-ray fluorescence and geophysical surveys. Results of the X-ray fluorescence survey, which were used to screen surface soils for the metals of concern (arsenic, chromium, and copper), were limited due to the relatively high instrument limits of quantitation for the analytes (81, 125, and 193 mg/kg, respectively). Surface soil concentrations for chromium and copper were not detectable. However, arsenic was detectable having elevated concentrations in several areas of the lumber yard and around the wood treating area. The geophysical survey was conducted using an electromagnetic conductivity instrument. Results from this survey were limited by cultural interferences from structures on the surveyed properties and overhead lines. Additionally, a shallow clay unit existed immediately below the site, which also affected the instrument's capability. These interferences resulted in a reduction in the planned geophysical survey.

In addition to the above-described investigative survey activity, an ecological survey and cultural resource survey were performed. The ecological survey provided information and observations to assess the status of the surrounding natural environment and to determine the potential ecological risk factor. As part of the ecological survey, various state and federal agencies were contacted to determine if any special status species or areas were within or adjacent to the study area. The information obtained from these agencies indicates that the Dismal Swamp southeastern shrew is a special status species that is potentially present within the study area. It is, however, unlikely to exist on the Saunders property.

A Stage 1A cultural resource investigation was performed. The investigation found the study area to have a high sensitivity for both prehistoric and historic cultural resources. At this time, it is recommended that the Virginia SHPO be contacted to inform them of the Saunders project, and to request their formal recommendations as to the type and level of additional cultural resources investigations that may be necessary.

The invasive portion of the site work focused on the following areas: surface soils, subsurface soils, groundwater, and surface water

and sediments. Surface soil investigation included areal composite sampling of the various operational areas of the Saunders and Kelly properties as well as discrete sample collection from areas of specific interest (i.e., the wood treating process area) to determine surficial contamination. The subsurface soil investigation was based on collection of samples through the unsaturated zone for the identification of below-grade contamination, and sampling into the underlying water-bearing zones to determine stratigraphy and hydrogeological characterization. Forty-three soil borings were installed. Of particular interest was that geologic units known to exist beneath the site were found at a much shallower depth (less than half) than the depth suggested by literature. These geologic units have been delineated as:

- An uppermost unit of fine- to medium-grained sand with isolated silty and/or marshy clay. This uppermost unit extends from the surface to a depth of approximately 12 feet across most of the Saunders property;
- A green-gray clayey unit approximately 2 to 7 feet in thickness is located beneath the uppermost sandy unit. This unit is identified as the confining clay layer in this report; and
- A silty sandy unit with locally fossiliferous bands was identified beneath the green-gray clayey unit. Regional studies indicate that this unit may be as thick as 100 feet at the site (Teifke 1973).

Hydrogeologically, the uppermost unit is defined as an unconfined aquifer of sands and silty sands (the Columbia aquifer). The lower unit is a confined aquifer of shell fragments, silts, and sand (the Yorktown aquifer). The middle unit is a confining clay layer of low hydraulic conductivity acting as an aquitard separating the two aquifers.

Groundwater gradient and contamination were evaluated through data collected from four existing site monitoring wells, two pumping wells (the Saunders recovery well and the irrigation well on the Kelly property), and 18 monitoring wells installed during this investigation. Ten of the wells installed during this investigation were screened in the shallow water-bearing zone and the remaining eight were screened in the deeper water-bearing zone. The deeper wells on the Saunders

property were isolated from the upper unit by double casing. Slug tests were performed on several of these wells to evaluate aquifer properties.

Surface water and sediment samples were collected from five areas to determine the extent of contamination. These areas include the existing wastewater pond, the intermittent stream, Godwin's Millpond, Chuckatuck Creek (outlet of the millpond), and the drainage swale, which discharges into Cedar Creek.

The following sections summarize the findings of the remedial investigation with respect to the nature and extent of contamination, fate and transport of contaminants, and baseline risk and ecological assessments.

10.2 NATURE AND EXTENT OF CONTAMINATION

Contaminants that appear to be distributed as the result of past wood treating operations on the Saunders site include arsenic, chromium, copper, PCP, and dioxin/furans. These contaminants have been identified as those of potential concern. Background or reference concentrations were determined using data collected from locations unlikely to be impacted by activities on the Saunders property. Cumulative frequency distribution plots were also constructed to evaluate background concentrations of arsenic, chromium (total), and copper in soils.

The results of the surface soil investigation suggest the distribution of contamination is throughout most of the Saunders property and a portion of the adjoining Kelly property. The widespread distribution of inorganic analytes reported above reference or background levels may be more extreme then actual on-site contamination because the surface soil sampling was biased to include any potentially elevated areas as indicated in the X-ray fluorescence screening. Soil borings were placed in areas of potential contamination based on past site operations. This biased sampling may exaggerate the actual extent of on-site contamination; nevertheless, elevated levels do exist, indicating that some degradation of site soil and subsequent water quality has occurred. Soil samples were not collected from beneath paved areas such as building floors or the concrete drip pad. Since wood-treating operations predate the paved areas, the paved areas particularly beneath the drip pad located in the wood treatment area may have elevated concentrations of some contaminants.

Most of the inorganic contamination is limited to the surface soils immediately surrounding the active wood treating operations, and the sediment in the runoff water catch basins. The surface soils and sediment in the runoff basins show elevated levels of arsenic, copper, and chromium. Unfiltered water samples collected from the monitoring wells within the unconfined aquifer downgradient and adjacent to the wood treating operations, and the intermittent stream west of the site show some evidence of arsenic, chromium, and copper contamination. Unfiltered groundwater samples were generally turbid, and analytical results obtained from these turbid samples may be more reflective of the sediments in the aquifer than the groundwater in the aquifer. Filtered groundwater samples indicated only the shallow monitoring wells immediately adjacent to the wood treating operations had elevated concentrations of arsenic and chromium with respect to the background concentrations. None of the concentrations detected in the filtered groundwater samples were above groundwater standards.

The primary organic contaminants include PCP and dioxin/furans. PCP contamination is limited mainly to soil and sediment on the Saunders property and groundwater. Most of the surface soil within the fenced portions of the property, and several isolated areas at greater depths, have concentrations of PCP that exceed 1,000 µg/kg. The sediments of the wastewater pond and the soil in the former earthen separation pond have the highest PCP concentrations. PCP in the groundwater is primarily limited to the shallow monitoring wells nearest to the wood treatment operation. PCP was also detected in one well screened exclusively in the Yorktown aquifer near the former conical burn pit area. PCP does not appear to have impacted the surface water or sediment of the intermittent stream or other surface water bodies near the Saunders property.

Low levels of dioxin/furans were detected in the background soil sampling and most of the sediment samples. Dioxins/furans were not detected in any of the water samples except for the water in the catch basins. Water from the catch basins was collected during a low flow period. This required the mounding of the sediments in the catch basins to pool runoff water for sample collection. Sediment from the catch basin was likely suspended in the water samples collected from these

catch basins. Elevated concentrations of dioxin/furans were detected in the runoff water in the catch basins, sediments of the wastewater pond, and areas around the former earthen separation pond and conical burn pit. Since burning activities were associated with the former earthen separation pond and conical burn pit, it is not unexpected that dioxin/furans are found in these areas.

10.3 FATE AND TRANSPORT

Past practice was to locate treated lumber staging areas directly on unpaved soils, allowing discharge of the PCP and CCA solutions directly to site soils. This practice provided a consistent source of contamination to the surface soils and possibly subsequent downward migration to the groundwater.

Past burning activities appear to have resulted in the creation of dioxin/furans, which were deposited primarily on the surface soils surrounding the burn areas. Estimated areal distribution of dioxin/furans and PCP material through burning activities was modeled. The observed high levels of dioxin/furans (as measured in TEFs) in the wastewater pond sediments and surface soil appear to be related to the areal distribution of the burn product.

Arsenic, chromium, and copper are still used in the wood treatment process. The operation has decreased its impact on the site by staging treated lumber on paved areas. Runoff from the paved areas is collected and used as treatment water.

The physical and chemical properties that tend to bind metals to soils has resulted in minimal downward migration of inorganic contamination, and most metal contamination is concentrated in the surface soils immediately surrounding the actual wood treatment area. The contaminants present in the surface soils can be suspended in the form of dust raised into the air by on-site vehicular traffic. Because metals tend to adhere to soils, little metal contamination is noted in the groundwater or surface water. Observed groundwater metal contamination is limited and of relatively low concentrations. Only the monitoring wells screened in the shallow soils with the highest metal concentrations show any evidence of metal contamination. Arsenic, chromium, and copper concentrations in the filtered groundwater from the site do not exceed established groundwater regulatory limits.

Surface water runoff from the site has the potential to entrain surface sediment. Since the surface soil has the highest concentration of inorganic contaminants of concern, it is not surprising to see relatively high arsenic and chromium concentrations in the runoff water. Analysis of samples collected from the catch basins on the east side of the site, which ultimately discharge to Cedar Creek, indicates that the relatively elevated levels of arsenic detected in the filtered surface water samples (9 to 15 µg/L) collected from the drainage swale, which ultimately discharges to Cedar Creek, may be the result of the runoff water from the site. Surface soil contamination from the western portion of the Saunders property has the potential to be discharged with runoff water off the property to the intermittent stream. Contaminants that move to the intermittent stream have the potential to impact the water quality of Godwin's Millpond. However, direct site-derived contamination was not evident in Godwin's Millpond and Chuckatuck Creek.

In addition to metals, PCP and dioxin/furans found in the surface soils also has the potential to be transported with the surface runoff water. Elevated levels of TEFs in the surface water runoff collected from the catch basins and elevated levels of PCP in the sediments from the wastewater pond confirm this transport pathway.

PCP and dioxin/furans tend to sorb to soils; however, downward migration may occur. Since PCP was used in a No. 2 fuel oil base, PCP, as a PCP/fuel oil mixture, may migrate downward through unsaturated soils as a nonaqueous phase liquid. PCP was found in the subsurface soils and in the groundwater. Separate nonaqueous phase PCP was not detected in collected groundwater samples, although the presence of isolated pockets of nonaqueous phase PCP liquid (either in the groundwater or vadose zone) cannot be ruled out in areas not sampled (i.e., under the drip pad). The clay layer between the two aquifers has provided a barrier to contaminant migration. However in areas where the integrity of the confining clay layer has been compromised (preexisting wells screened across both aquifers), the potential exists for downward migration of organic contaminants to the lower confined aquifer. PCP detected in one lower aquifer well near the preexisting wells suggests that this transport has occurred.

10.4 BASELINE RISK AND ECOLOGICAL ASSESSMENT

Based on the information and observations and analytical data gathered by the investigation, baseline risk to human health and the surrounding ecological environment was assessed.

Both worker and residential risks were evaluated. Under current land use conditions, estimated cancer risks for workers by all three soil exposure routes--ingestion, dermal adsorption, and particulate inhalation--exceeded the benchmark level of 10^{-6} as recognized by EPA Region III. Similarly, under potential future on-site residential use conditions, cancer risks for all three soil exposure routes also exceeded the 10^{-6} benchmark. Arsenic and dioxin/furans soil concentrations are the dominant contributing factors to the cancer risks for all three soil exposure routes for both land use conditions.

Estimated cancer risks for residential groundwater usage, both lower and upper aquifers, also exceeded the 10^{-6} benchmark.

None of the exposure pathways under current land use conditions were estimated to result in potential exposures to noncarcinogenic contaminants that might produce adverse health effects. However, potential exposure to noncarcinogenic contaminants could result in adverse health effects should the Saunders property be converted to residential use without prior remedial measures, if the groundwater from the upper unit were to be used as a residential water supply. The hazard index for the upper aquifer was 12. The hazard index benchmark is 1. This condition is highly unlikely due to the limited groundwater yield of this unit. Utilization of the lower unit, which has a hazard index of less than 1, would not produce non-carcinogenic effects.

The ecological assessment has found evidence suggesting a potential for adverse ecological impacts in sediments of Godwin's Millpond and adjacent intermittent stream aquatic habitats, based on laboratory toxicity tests and the paucity of aquatic macroinvertebrates. However, the spatial extent of this contamination indicates a source(s) other than Saunders. The spatial distribution in surface waters and sediments does not provide any evidence that contaminants related to Saunders are the causal agent of adverse ecological impacts. The status of aquatic life in the area suggests that the health of the natural habitat is good and that the suggested contamination may only be moderately affecting

the ecosystem. Fish populations of recreational importance appear to be adequate despite the observed contamination. Observations of the terrestrial ecosystem also appear to be adequate and do not show an indication of adverse impacts.

10.5 CONCLUSIONS

The RI has shown that activity on the Saunders property has contributed to the contamination of site soils and groundwater beneath and immediately adjacent to the Saunders property. The extended surrounding environment, particularly Godwin's Millpond and Chuckatuck Creek, appears not to be directly affected by contamination emanating from Saunders. The impact observed on these systems appears to be a result of all land use activity within the watershed area of the millpond and creek.

The presence of contamination on the Saunders property currently creates a potential health risk to the workers and impacts future use of the property should it be considered for residential development. Also the surface drainage to the east empties into a drainage swale of Cedar Creek, hence extending possible current risk beyond the immediate site boundary.

It is recommended that the FS phase of the RI/FS process be conducted to determine remedial alternatives for current and future reduction of risk. Data limitations of this investigation and guidance toward recommended remedial action objectives are presented below.

10.5.1 Data Limitations and Recommendations for Future Work

Sampling methodologies and locations were selected based on the best available data generated by previous site investigations. Where necessary adjustments to actual field conditions were made in agreement with EPA Region III staff and duly noted. All analytical data was subjected to data validation in accordance with EPA guidance.

The areal distribution of contaminants in the surface soil may be overestimated due to the sample compositing within each area. Although this method of sample collection has identified certain areas of elevated contamination, it does not specifically locate where a concern exists. Thus, depending on the extent of possible remediation, additional sampling may be appropriate within the areas of concern and in

adjacent properties to more definitively identify contamination. Additionally, past cut and fill activities on the site have obscured the distribution of contaminants in site soils. These activities include but are not limited to the rerouting of unimproved roads, building construction, changes in the treatment process, elimination of the earthen separation pond and the former conical burn pit, and the creation of the wastewater pond. These conditions must be considered when evaluating remedial alternatives to determine the extent of verification sampling versus unit costs for soil remediation to assure that the cost of sampling does not exceed the cost of remediation.

The monitoring wells indicate that most of the groundwater contamination is limited to the area immediately adjacent to the wood treating operations. PCP was the primary contaminant of concern detected in the monitoring wells. Only one well screened entirely in the lower aquifer unit had concentrations of PCP above the MCL of 1 µg/L. This well was located very near to the wells installed in earlier investigations. It is possible that the recovery well installed to recover PCP actually accelerated PCP movement through the clay by lowering the potentiometric surface of the lower aquifer unit, increasing the head of the unconfined aquifer and facilitating downward groundwater and contaminant migration through the clay. The preexisting wells are not known to have been properly sealed and should therefore be plugged to prevent direct communication between the two aquifer units.

Future considerations for any additional sampling should be tailored to address data gaps that must be bridged to effectively treat the observed contaminant distribution. An example would be to collect successive samples of the clay layer to be analyzed for PCP to evaluate if PCP is migrating through the clay layer. If the PCP is rapidly moving through the clay layer, then more than plugging of the existing wells will be required to prevent leakage of contaminated groundwater across the clay. To more effectively determine the partitioning of PCP between the aquifer materials and the groundwater, saturated soil samples should also be collected near the monitoring wells, analyzed for PCP, and compared with groundwater PCP concentrations. Turbid water samples distort reported contaminant concentrations. If the monitoring wells cannot be adequately developed, then filtered samples should be collected.

10.5.2 Recommended Remedial Action Objectives

Based on the results of this investigation, the development of remedial alternatives (to be performed as part of the FS) should consider corrective alternatives for arsenic, PCP, and dioxin/furans contamination in site surface soils and surface runoff; PCP and dioxin/furans contamination in site subsurface soils and sediments of the wastewater pond and storm sewers; and PCP contamination in groundwater.

Anticipating the potential for site remediation, several treatability alternatives for soil and groundwater were screened during this investigative phase. Incineration was found to be an acceptable technology for soil remediation. The leachability of PCP was also evaluated under laboratory conditions simulating natural soil percolation. PCP was found to be leachable, but most likely will be influenced by soil type, requiring specific solvent types to be determined. Biotreatability tests were performed on soils obtained from the Saunders property. From the collected soils a bacterial culture capable of degrading PCP was developed. However, attempts to cultivate the bacteria in moderately contaminated soils collected from the Saunders property failed. The screening study indicates that biodegradation may be ineffective at the Saunders property, although additional studies would need to be performed to confirm this. Groundwater evaluation was limited to carbon adsorption tests for the removal of PCP. This treatment alternative was found to be acceptable. Biotreatability of groundwater was considered but not conducted due to the low concentrations of PCP detected in the groundwater.

As part of the FS process, consideration must be given to the potential presence of the Dismal Swamp southeastern shrew, and both prehistoric and historic cultural resources that may be impacted during remedial investigations.

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UNSCANNABLE ITEM

SITE NAME Saunders Supply Co.

OPERABLE UNIT

ADMINISTRATIVE RECORDS- SECTION III VOLUME C

REPORT OR DOCUMENT TITLE Remedial Investigation Report

DATE OF DOCUMENT

DESCRIPTON OF IMAGERY Site facilities and Topographic Map

NUMBER AND TYPE OF IMAGERY ITEM(S) 1 oversized map

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REPORT OR DOCUMENT TITLE Remedial Investigation Report

DATE OF DOCUMENT

DESCRIPTON OF IMAGERY Geophysical Investigation Terrain Conductivity Traverse Line Location Map

NUMBER AND TYPE OF IMAGERY ITEM(S) 1 oversized map